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DISEASES

BY

PROFESSOR OF DISEASES OF THE MIND AND NERVOUS SYSTEM AND OF MEDICAL JURISPRUDENCE,
MISSOURI MEDICAL COLLEGE, ST. LOUIS; LATE PHYSICIAN IN CHIEF TO ST. VINCENT'S
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1892.

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1892

THIS WORK
IS RESPECTFULLY DEDICATED
TO
J. M. DA COSTA, M.D., LL.D.,
EMERITUS PROFESSOR OF PRACTICE OF MEDICINE AND OF CLINICAL MEDICINE
AT JEFFERSON MEDICAL COLLEGE, PHILADELPHIA,
BY HIS
FRIEND AND FORMER PUPIL,
THE AUTHOR.

PREFACE TO THE SECOND EDITION.

THE first edition of this work was published in 1876, and has been exhausted for many years.

The author now submits this new edition at the repeated solicitations of his former pupils, an exacting practice having prevented an earlier compliance with their requests.

No thought nor labor has been spared in the effort to place the subject-matter of these pages on a line with contemporary neurology, which has of late years advanced by unexampled strides.

The lectures have been entirely rewritten, and the author hopes that they will be favorably received as a critical analysis of the matters dealt with from the stand-point of a long, studious, and extensive experience.

Special efforts have been made to present fairly both aspects of unsettled questions, and at the same time to render the work as comprehensive and practical as possible.

Anatomical detail and physiological discussion have been thought inadmissible, partly on account of the practical aim of this work, but more especially because so many standard works of reference are accessible in which such subjects are elaborately treated.

The consideration of insanity in these lectures has suggested the propriety of dividing them into two parts. In the second volume diseases of the brain and of the spinal cord, and functional and peripheral affections of the nervous system, will be discussed.

The author desires to express his grateful acknowledgments to his esteemed and erudite friend Dr. W. H. Ford, of this city, whose advice and assistance have been invaluable in the completion of this undertaking.

2808 OLIVE STREET, ST. LOUIS, January, 1892.

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DISEASES OF THE NERVOUS SYSTEM.

LECTURE I.

THE CEREBRAL CIRCULATION.

GENTLEMEN,—Variations of the quantity of blood in the brain may take place under many conditions. The cerebral substance is enveloped by three membranes. The dura,* which is outermost, furnishes certain folds or prolongations to form the so-called cerebral sinuses, which are lined by the continuation of the serous membrane of the veins, and convey the venous blood from the brain. The innermost membrane, the pia, is the vascular membrane, and from it most of the arterial supply finds its way into the substance of the brain by the capillary vessels. The intermediate membrane is the arachnoid, which is reflected upon itself, the space between the folds constituting its cavity. This latter membrane does not, like the pia, follow the surface of the cerebrum into its depressions or sulci, but, on the contrary, stretches across the depressions, and leaves between itself and the pia a space called *subarachnoid*, which contains a liquid,—the cerebro-spinal fluid. The amount of this fluid in the cranial cavity is not constant, but is in inverse ratio to the amount of blood in the vessels.

In this connection the student should remember that the *deep lymphatics of the cranium*, according to Gray, consist of two sets, the meningeal and the cerebral.

* At a recent congress of European neurologists it was decided, among other changes in nomenclature, to drop the word *mater*, using simply the words *dura* and *pia*.

"The *meningeal* lymphatics accompany the meningeal vessels, escape through foramina at the base of the skull, and join the deep cervical lymphatic glands. The *cerebral* lymphatics are described by Eshmann as being situated between the arachnoid and pia, as well as in the choroid plexuses of the lateral ventricles: they accompany the trunks of the carotid and vertebral arteries, and probably pass through foramina at the base of the skull, to terminate in the deep cervical glands. They have not at present been demonstrated in the dura or in the substance of the brain."

The perivascular canals are *conduits* formed by the pia around the vessels of the brain.

The perivascular lymphatics are lymphatic vessels or plexuses ensheathing blood-vessels.

The perivascular sheath is a sheath of pia forming a perivascular canal.

The perivascular spaces, or "Virchow-Robin's spaces," are lymph-spaces between the outer and middle coats of an artery.*

In the study of cerebral circulatory disturbances, the influence of the perivascular spaces is not so clearly understood as we might wish. That they and their contents participate in the phenomena of congestion cannot be doubted.

A greater flow of blood in the arterioles is necessarily accompanied by increased afflux in the above-mentioned conduits, with corresponding augmentation of the amœboid movements.

This view is corroborated by Virchow's assumption "of the existence of *diffusive currents* (endosmosis and exosmosis) between the contents of the vessels and the fluid in the tissues; and by regarding the capillary wall as a more or less indifferent membrane, forming merely a partition between two fluids, which enter into a reciprocal relation with one another; while the nature of this relation would be essentially determined by the state of concentration they are in and their chemical composition, so that, according as the internal or the external fluid was the more concentrated, the diffusive stream would run inwardly or outwardly, and according to the chemical peculiarities of the individual juices, certain modifications would arise in these currents."

In this connection I must again quote Virchow, to the effect

* For these definitions see Billings, Nat. Med. Dict., 1890.

that "we must not, however, go so far as to ascribe to this membrane all the peculiarities observable in the interchange of material, and so explain how it happens that certain matters which enter into the composition of the blood are not distributed in equal proportion to every part, but leave the vessels at some points in greater, at others in less quantity, and at others not at all. These peculiarities depend, manifestly, on the one hand, upon the different degrees of pressure to which the column of blood is subjected in certain parts, and, on the other, upon special properties of the tissues; and we are irresistibly compelled, both by the consideration of simply pathological, and particularly by that of pharmaco-dynamical phenomena, to admit that there are certain *affinities* existing between definite tissues and definite substances, which must be referred to peculiarities of chemical constitution, in virtue of which certain parts are enabled in a greater degree than others to attract certain substances from the neighboring blood."

I would observe, therefore, that, notwithstanding the usual contents of the perivascular spaces, at times, under the influence of circulatory disturbances, more or less cerebro-spinal fluid will be directed thereto: hence the philosophy of the cerebral circulation would not be complete without a reference to their relationship to it.

Gowers, in his "Diseases of the Nervous System," observes upon this subject, "It was at one time thought that the blood in the brain could not vary in amount, because the cranio-vertebral cavity is a closed space, and this opinion is still occasionally put forward. *But the mobility of the cerebro-spinal fluid (which occupies not only the inter-membranous space and the ventricles, but also the lymphatic spaces around the vessels)** permits the vascular distention to vary. If the cavity were hermetically closed, the variation could be only relative, not absolute. But the numerous foramina of the cranium and vertebral canal are occupied by less resistant structures, which no doubt may yield in some degree. Moreover, the large surface veins of the spinal cord, and still more the enormous plexus outside the spinal dura, doubtless constitute an important means of adaptation. Further, the

* Italics my own.

processes of secretion and absorption of the cerebro-spinal fluid, always in constant operation, must be influenced by the degree of pressure, and may quickly vary with it. Although the conditions during life and after death are widely different, yet we may reasonably regard the enormous variations in the total amount of blood within the cranio-vertebral canal after death in different cases as evidence that considerable variations may occur during life. Some variation is physiological. In the child, before the fontanelles are closed, and in the adult when a piece of the skull is removed,* it is seen that the brain pulsates synchronously with the heart, and that variations also result from the respiratory movements of the thorax. Tracings of these movements have been obtained."

Tuke † thinks that "the importance of the lymphatic system of the encephalon as a factor in morbid processes has been too much overlooked."

In connection with this subject, and in corroboration of the views I have presented, I quote the following observations from a thesis sustained before the Faculté de Médecine of Paris, entitled "*Contribution à l'Étude de l'Encéphalocèle acquise*," by Dr. Lewis A. Lebeau, of St. Louis, in 1875: "In the normal condition the brain is contained in the cranial cavity and suspended, as it were, in the midst of the cephalo-rachidian liquid, which fills the void which occurs when the sanguineous afflux diminishes in the nervous centres; the different parts of the encephalic mass are enclosed in the folds of the dura mater, and cannot therefore be compressed; the entire mass, it is true, is enclosed in an incompressible cavity, but a part of its contents can escape when cerebral congestion occurs; there exists a constant relation between the quantity of blood which enters therein with every cardiac systole and the cerebro-spinal fluid correspondingly displaced. At the same time it is important to remember that there exists the passive stasis of blood in the cerebral veins during forced expiration, and during all muscular efforts associated with the physiological efforts which excite these respiratory acts.

"There is an embarrassment of the returning circulation, the

* "In an operation that I recently witnessed (removal of a tumor from the spinal cord), before the dura was opened its distention with every movement of respiration was most conspicuous."—*Gowers, Dis. Nerv. Syst.*, ed. 1888.

† *Ann. Univ. Med. Sci., Sajous*, 1891.

blood in the jugular veins is prevented from emptying into the right auricle and produces a mechanical obstacle to a free flow of the blood in the cerebral veins, pronounced stasis and turgescence therefore follow ; in both cases there is a corresponding augmentation of the volume of the brain, but in the first instance the arterial pressure is not continuously sustained, but the cardiac pulsations do not cease. In the second case there is a mechanical engorgement which persists as long as the pulmonary cause which produces it lasts. Venous stasis of the brain is very readily produced, as the jugular veins have insufficient valves ; in this connection it should be stated that Mr. Guyon considers that the thyroid gland exercises a compression upon these vessels, thereby preventing an increased flow of blood to the brain, and consequently diminishes the engorgement of that organ.

"Messieurs Pelletan and Bourgougnon adopted the extreme view, in consideration of the fact of the incompressibility of liquids, that the brain does not pulsate, notwithstanding the unquestionable existence of arachnoidean pulsation. Schlichting, Lorry, Lamure, in their memoirs in the Academy of Sciences, and Haller, in his *Opera Minora*, recognized and demonstrated the incontestable pulsation of the brain. Moreover, Monsieur Richet taught, in his work on Surgical Anatomy, that a continual afflux and reflux of the cerebro-spinal fluid existed between the cranial and arachnoidean cavities ; he, moreover, claimed that the rachidian cavity was to a certain extent an 'escape-tube' which permitted the brain to become engorged, and just in proportion as its volume is augmented the more will the liquid in the spinal canal be expelled ; moreover, the cerebro-spinal fluid is never the same in quantity : its quantity varies from day to day and accumulates in proportion to the space existing in the encephalic cavity.

"Nevertheless, the cerebral engorgement does not pass a certain limit ; when in the physiological condition the maximum cerebral congestion is attained, there nevertheless remains a small quantity of the cerebro-spinal fluid between the brain and the cranial bones ; but this condition is not the same in pathological cases ; the penetration of air into the midst of the arachnoidean cavity prevents the approximation of its respective folds, capillary circulation ceases, and the cerebro-spinal fluid flows towards the base of the

brain ; in fact, in no case is the presence of this fluid noticed in fractures of the vault of the cranium ; on the other hand, it is one of the signs of fractures at the base of the skull ; the local afflux of arterial blood is thus augmented, and it is easily comprehended that there may be a more or less considerable engorgement at the points where the osseous structures have yielded, and, in consequence of the little resistance of the brain at any point of the cranial vault, the cerebral congestion is transmitted to the pia mater, finally to the cranial bones ; these conditions are somewhat relieved by the reflux of the cerebro-spinal fluid."

Some have maintained that the brain, being tightly held in the closed cranial cavity, will not admit of any variation in the quantity of the blood therein contained unless there should occur an inverse variation in the quantity of its solid contents. In contradiction to this opinion, the theory has been advanced that the cranial cavity is not absolutely closed, and that the quantity of blood circulating in the brain may be augmented by cerebral compression, the result of which would necessarily afford increased capacity, with corresponding reduction of volume.

Were the cerebral substance strictly compressible, the amount of cerebro-spinal fluid might actually remain constant ; for when a larger amount of blood than usual found its way into the brain, its solid constituents would simply be forced to occupy a smaller space.

This, however, is not the fact ; and although, perhaps, by pressure, you might bring the several particles of the brain into closer proximity, you could not, without causing molecular disintegration, make them occupy less space than previously.

The brain may be compared, as was cleverly suggested by Dr. Elam, to a sponge filled with fluid. By firm pressure you can cause all of the latter to escape from the porosities of the former. You can make the sponge apparently smaller than it was before the fluid was expressed, but it is impossible to reduce its volume absolutely by any pressure you may exert. The pores which existed in its substance, it is true, have been obliterated ; but the constituent particles of the solid have by no means been compressed into a smaller space, which could be accomplished only by their actual encroachment each upon the other, an occurrence which, if possible, would doubtless lead to their ultimate disor-

ganization. Therefore, as Dr. Elam clearly shows, the human brain is certainly incompressible. *Dynamically* speaking, however, a pressure may be exerted materially influencing this supreme nerve-centre, not by a reduction of its size, but by producing unmistakable effects upon its molecular structure through the active circulation of blood when flowing in undue quantities, and under a variety of pathological conditions, in its delicate and highly-organized substance.

Hence vascular tension, occasioned by fluctuations in the quantity of the cerebral circulation, becomes an important factor in the explanation of many of the phenomena which we have to study.

We can readily understand that fluxion and stasis may eventuate in molecular changes profoundly influencing the interstitial nutrition of an organ so remarkably sensitive and impressionable, in consequence of the activity of its physiological tissue-metamorphoses. This blood-pressure may be better understood if we consider Dr. Elam's illustration, which most forcibly struck me in reading his valuable book. He states that "glass is sufficiently incompressible to be considered altogether so ; yet glass may be subjected to pressure, and the effects upon its molecular structure are very striking and suggestive. Even the slight pressure that may be exerted by the fingers is sufficient very materially to alter its optical properties, especially as to its relation to polarized light ; and the change continues so long as the pressure is continued."

It may, in conclusion, be safely affirmed that, while the quantity of *blood* circulating in the cranial cavity may undoubtedly vary, the sum total of all *liquids present* is constantly and uniformly the same ; which fact we have already stated is explained by the inverse ratio of compensation existing between the vital current and the cerebro-spinal fluid.

In a word, we may unequivocally maintain that in order to change the relative amount of cerebro-spinal fluid we must have an increased amount of blood in the brain.

The blood contained within the cranial cavity is thus susceptible of notable variations of its volume, while in quality it may be affected by a host of systemic conditions.

The cerebral circulation plays a most important rôle in the study of cerebral congestion, and the latter in all conditions in

which as a *primary* factor, whether active or passive, it produces serious disturbances, with an accompanying train of morbid phenomena. *Secondary* congestions—a result of a multitude of varying pathological cerebral lesions—are of no less interest, and offer the only reasonable interpretation of numerous fluctuating symptoms and daily mutations in the condition of patients affected with cerebral disease. This peculiar variation of symptoms, the daily alternation of those of excitation with those of depression, the appearance and disappearance of the disturbances of motility within a few hours in certain cases, may be thus explained satisfactorily.

The views of Schroeder van der Kolk upon the circulation of the brain are, I think, exceedingly important, and I therefore proceed to quote, on this subject, from his work on the Pathology and Therapeutics of Mental Diseases :

“To rightly understand the lesions of the brain in mental diseases, we must keep in mind the relation of the circulation to the vitality of the brain. It is evident that variations of intravascular pressure of the blood, and of its arterial or venous conditions, as well as other modifications of it, cannot remain without important influence upon the brain.

“Daily experience teaches us that violent congestion of blood may interrupt the functions of the brain, and speedily lead to a fatal termination.

“When by an increased activity of the heart the blood is more powerfully and under greater pressure driven into the aorta, it runs off for the most part and quickest through those vessels in which the least resistance and counter-pressure have to be overcome.

“Let us now remember some of the branchings of the great vessels, which are quite remarkable. In the first place, we must recall the fact that the inferior thyroids rise close to the vertebrae, and that at the division of the common carotid into the internal and external carotids the superior thyroid is given off. There is undoubtedly a definite purpose to be attained in this doubling of the thyroids on each side : it is not merely to secure a more copious supply of blood to the thyroid gland, for the latter object could have been very readily reached if either of the two thyroids had received a greater calibre. The thyroid gland can take up a large

quantity of blood, and is capable of great expansion, since Forneris claims to have ascertained by measurement that in consequence of its expansion the neck is larger during sleep and on awaking than during the day. Also in the dead body the size, the consistence, and the vascularity of this gland differ remarkably. We may, then, assume that the propelled blood finds less resistance in the two extensile thyroids than in the vertebrales and internal carotids, which, on account of their course through long canals, can increase only slightly in diameter and not at all in length, and on that account it flows off in greater quantity towards the thyroid gland. Therefore, with its other functions, the thyroid may be regarded as a diverticulum or reservoir by which a too strong pressure of blood may be diverted from the brain. The above-described arrangement of these arteries is, moreover, not limited to mammalia. According to J. Simon, the thyroids of the bird arise exactly opposite the place where the carotids and vertebrales come off, and even in amphibia and fishes these vessels stand in relation with the vessels of the brain.

“From this it becomes not improbable that the presence of a goitre, which receives a great deal of blood and which may derive too powerfully from the brain, induces in cretins a weaker development of the brain, or, at least, a debilitated energy of it; although cretinism is not to be regarded as a product of goitre, but is frequently concomitant with it. It is well known that individuals afflicted with goitre are frequently dull and phlegmatic. It is probably also for the same reason that sometimes in meningitis an atrophied thyroid gland is found, because through this condition the derivation of the blood-stream from the brain would be impeded, which then led to repeated congestions, and contributed to the development of meningitis.

“The spreading of the vessels in the face conduces to derivation from the brain. There are few other arteries in the body so readily filled with blood as the above, because they are everywhere surrounded by soft fat, and on that account the coats of the vessels have no powerful support from without. Thence it occurs that the cheeks, and indeed the whole face, so easily assume an increased color in powerful and sudden emotions, especially also from high degrees of temperature. If the vessels of the brain were as readily filled, apoplexy from every active exertion

might be imminent. The external carotid artery thus leads the strong pressure of blood outward, because the internal carotid in the closed cavity of the skull cannot equally well expand, and on that account opposes a more powerful resistance to the blood-pressure. We cannot always conclude from diffused redness of the face that there is congestion of the brain, although the latter is frequently associated with the former.

[Schroeder van der Kolk, in a foot-note, here states that the researches of Suequet corroborate these views. He has demonstrated, in addition to the proper capillaries, larger communicating branches between the arteries and veins in the hand and elbow, in the foot and knee,—even in the face, in the skin of the lips, nose, eyelids, eyebrows, and ears, and in the mucous membrane of the cartilages and septum of the nose. He correctly claims for these anastomoses a derivative purpose, as in strong arterial pressure the blood will be carried on more quickly through them towards the veins. In old age these communicating branches increase in number and size.]

“Similar conditions occur with the vertebrales. These arise from the subclavians, and with strong pressure of blood the stream can be derived from the vertebrales towards the arm. For this reason may hand-baths, or placing a bandage around the arm or even the finger, stop bleeding from the nose: this I have many times experienced with the best effect, and in earlier years was accustomed to verify upon myself.

“The resistance against a strong blood-pressure is still more increased by the enclosure of the brain and spinal cord in bony cavities, which are not capable of extension, and which under ordinary circumstances must always contain the same volume of solids and fluids, as no elastic structure occurs in them. Hence it has been assumed that generally no increased quantity of blood can proceed to the brain, as the latter is not capable of compression. But in apoplexies we often find large quantities of extravasated blood.

[Schroeder van der Kolk here states that he had in his possession a preparation where the blood was extravasated in so great quantity between the dura and the skull that the coagulated mass of blood, on opening the skull, was bigger than the fist, and had pressed the hemisphere quite flat. The blood was freshly coagulated; its extravasation had occurred but recently, and with great rapidity, so that death must have been immediate.]

“Therefore the liquor cerebro-spinalis can afford room for a larger quantity of blood, while it escapes out of the cavity of the

skull into the more lax and movable sac of the dura mater spinalis, which in the natural condition does not appear to be full and tense. For if we carefully open the vertebral canal so that the dura mater is not injured, the sac may be inflated to a larger size. For, although the skull-cavity is not thoroughly unalterable, the blood-vessels have yet a stronger support from it, and it opposes a stronger resistance to too great filling with blood. But a certain scope is indispensable, as every exercise of power, and consequently also activity of the brain, demands a more active circulation and increased change of material, on which account a powerful and continuous mental exertion occasions phenomena of congestion, and at last induces a feeling of heaviness, dulness, and fatigue.

“Kellie claims the discovery that, on account of closure of the skull, the blood is retained in its cavity, and therefore, after fatal hemorrhage, the brain does not appear so bloodless as the other parts of the body. If, on the other hand, through preliminary trephining he permitted the entrance of air into the cavity of the skull, he found the brain also bloodless. Dieckenhoff, however, could not corroborate this statement, and I have myself found the brain quite pale and bloodless in rapidly-fatal hemorrhages. Nevertheless, it appears to me that Kellie's assumption is not entirely to be rejected. For the most part we find the brains of slaughtered sheep not quite bloodless. But if the vessels of the brain become emptied through hemorrhage, the space must be filled by something else, and, according to Kellie, it is by serous exudation. We may, however, assume that in a rapidly-fatal hemorrhage the serous fluid runs from the spinal canal into the skull-cavity, and supplies the place of the diverted blood. The space forming in the spinal canal must then be balanced by filling and distention of the wide *venæ spinales*, which veins communicate with the *venæ cephalicæ* and stand in reciprocal relation.

“Nevertheless, the blood will be retained, if not completely, yet longer in the brain than in any other organ.

[Schroeder van der Kolk says that in post-mortem examinations, when the existence of brain-congestion becomes a question, we must bear in mind the suction-power exerted on the blood. The brain and brain-fluids contract on cooling of the body, and the blood in the veins, which still remains fluid

several hours after death, is partly driven back to the brain in order to fill the space formed through its shrinking. Nasse has already pointed this out. Thence also may it arise that the blood in the veins of the brain is mostly not coagulated, because the fibrin remains behind, and only the fluid blood returns into the cavity of the skull; therefore, in sections, the filling of the vessels of the brain gives no accurate measure of their condition during life.]

“In lessened blood-pressure, after copious loss of blood, a more active exudation of serum may easily occur; and this explains why abstraction of blood operates so deleteriously in the insane, especially in melancholic patients. If serum is once exuded, it opposes a more active circulation, the brain remains, therefore, longer anæmic, and softening of the brain and imbecility may be induced by the exuded serum. This frequently happens in such cases after venesection.

“Strong pressure of blood, resulting in distention of the vessels of the brain, as is often the case in chronic insanity, does not preclude the passing off of the superabundant blood in other ways. If the blood in the skull-cavity is under a stronger pressure than in the face, it has an outlet through the ophthalmic arteries, and the nose and the supraorbital region through the frontal artery acquire a deeper color. If the congestion has a more chronic course, as in many cases of acute mania, then only the tip of the nose is more deeply colored. But the same also occurs in melancholic patients. Hence we also observe in confirmed tipplers, in whom for the most part habitual brain-congestion exists, a red or bluish swollen nose. As the ophthalmic, through the anterior ethmoidal artery, extends along the septum narium as far as the tip of the nose, and likewise the skin of the nose externally is in connection with twigs of the ophthalmic and angular, we may easily comprehend how it is that the ophthalmic is diagnostically so significant, if the blood sustains a stronger pressure in the cavity of the skull and partly flows off through the ophthalmic.

“We have in the color of the sclerotic an entirely uncertain sign. Its vessels appear, in cerebral irritation, sometimes to contract under the influence of the ciliary nerves; at least, I have myself observed the sclerotic still white in very intense cerebral congestion of several days' duration, and in apoplexy.

“Bleeding from the nose is a favorable event in congestions of the brain. If it does not occur, we apply a leech in the nose, from which I have several times seen the best results.

[Our author states that he has frequently injected the ophthalmic alone in the skull-cavity. The forehead, the tip of the nose, and the cheek were colored thereby.]

“The vertebral arteries before they enter the cavity of the skull give off muscular branches which communicate with the posterior auricular. It is due to this fact that children frequently before convulsions have the ears reddened. In some maniacal patients the nose is colored, and in others the ears, which is to be explained as follows: in the first the congestion occupies the anterior portions of the brain, in the latter the posterior portions. It is for this reason that in congestions and in epilepsy repeated cuppings in the neck act favorably.

“The deeper color of the lower eyelid acquires a decided diagnostic value as it occurs, for example, in the climacteric years, or sympathetic with uterine diseases; especially is this the case when other diagnostic signs of *venous* congestion occur.

“It might appear as if the firmness of the vessels of the brain were impaired by their walls being thinner; but the walls of the vessels seem to be thinner rather for the purpose of avoiding rupture of the capillaries. The middle muscular coat is almost entirely wanting; through its contraction the blood experiences a stronger pressure, and is driven into the smaller vessels; but, on the other hand, the inner and outer coats of the vessels afford to the latter their proper firmness and power of resistance. From absence of the muscular coat, the walls of the vessels yield more easily to the stream of blood, offering less resistance to it, and also driving the blood with less force through the capillaries, which in the brain are so delicate and receive so little support from without. The consequence of this is a more even, not pulsatory, stream of blood in the capillaries of the brain. For, while the larger vessels which form numerous anastomoses run a considerable distance on the pia mater, although on account of their easy distensibility they readily yield to each stroke of the pulse by reception of the inflowing blood-wave, no pulsation is conveyed to the smaller vessels of the brain by them. In this, also, may be sought the significance of the *rete mirabile* which occurs in the skull-cavity of many mammalia,—namely, in the ox, sheep, and deer.

“The pia mater covers the cerebral convolutions, and from its

under surface very fine capillaries pass into the gray substance, where they inosculate freely and then pass into fine veins, which again return to the pia mater, and here unite into larger branches. Now, if the blood in the vessels of the pia mater, in order to pass into the veins, must all go through the cortical layer of the convolutions, every more active determination of blood, for example, even in any violent movement, must immediately manifest itself in so easily excitable a cortical layer. But that is not the case, and for this reason, that in the pia mater itself a free communication exists between the arteries and the veins, of which I have convinced myself by means of injections.

"Thus, in a violent congestion the blood for the most part passes away over the cortical layer, without acting on this, into the veins; the storm, as it were, sweeps away over us without our perceiving it. However, the effect of such a congestion always makes itself known.

"The cells in the cortical substance are at the moment in a condition of too great excitement; the ideas and images chase one another, and are not under control; deeper reflection requires a previous calming of the circulation. If the rapidity and strength of the circulation increase still more, as sometimes in fever, then delirium may be reached; the involuntary ideas and images gain in strength, so that they are no longer to be distinguished from real impressions.

"Further, due regard should be bestowed upon the sinuses of the dura mater, which are not capable of distention. If by impeded respiration the outflow of blood from the jugular veins into the chest is hindered, the accumulation of blood in the skull nevertheless meets a resistance, on the one side from the closure of the skull itself, on the other because the sinuses cannot expand; by means of the numerous anastomoses between the *venæ spinales* and the *azygos*, the blood can then collect more in the abdomen.

"The brain floats in the cerebro-spinal liquid almost as in a bath, so that, according to the researches of Poltz, it presses on the base of the skull with only one-fiftieth of its weight. Remember that the arteries, which on account of their power of expansion can endure a greater pressure, all lie on the base of the skull, but the easily-compressed veins are collected on the surface of the hemispheres, and have their position mostly in furrows between the

cerebral convolutions, so that they are not compressed by turgidity of the brain, and consequently the flow of blood back from the brain remains as free as possible. Moreover, between the branches of the arteries, as well as between the veins, numerous anastomoses are everywhere found, and with increased flow of blood no violent congestion of the brain can occur, because the pressure is divided equally on all the arteries and veins. Without this disposition a stronger flow of blood through one of the cerebral arteries would immediately have as a consequence a stronger congestion in a particular section of the brain, through which dangerous extravasations of blood would easily arise. Now, also, when from any cause the flow of blood is cut off at one place, the blood may pass through lateral branches, so that the circulation still continues. Nevertheless, we see, in embolism of the vessels of the brain, that such an obstruction may be very deleterious as soon as it extends over a rather large region.

“Through all these harmoniously-combining causes it becomes possible that the brain with its vessels, notwithstanding the delicate structure of the latter, is in a position to sustain a considerable pressure. We see this in difficult parturition, in asthma, in whooping-cough, in epilepsy, where the small vessels in the loose tissue of the eyelids, which arise from the ophthalmic coming out of the skull, burst in consequence of the great congestion of blood, without any extravasation occurring in the brain itself.

“We owe to A. H. Durham some valuable observations on the circulation during sleep. It was formerly supposed that in sleep a larger quantity of blood accumulated in the brain, and that increased venous congestion caused the coming on of sleep. This view was favored by increasing sleepiness in plethora, and also by the accumulation of blood in coma, and by the occurrence of sleep after an epileptic attack, where evidently congestion is present, and not less the state of unconsciousness in apoplectic effusions or in capillary injections of the vessels of the brain. But we must clearly distinguish tranquil sleep from coma and from sopor. A portion of the vault of the skull was removed from a dog with the trephine, and then the underlying dura mater excised; the uncovered part of the brain appeared to press into the opening, the large veins on the surface were somewhat distended, the smaller vessels of the pia appeared to be full of

dark blood, and no decided difference of color between arteries and veins could be perceived. Such was especially the appearance and condition during the action of chloroform. After the action of the chloroform had ceased, the animal fell into a comparatively natural and sound sleep; thereupon the surface of the brain became pale, and sank rather below the level of the bone, the veins were no longer distended, little vessels having an arterial color could be distinguished, and many which before swelled with dark blood could no longer be recognized. When after some time the animal was awakened, a weak red color appeared to spread over the surface of the brain, and the latter again pressed into the opening in the bone. The more active the dog was, the more the pia became injected, and the more turgid was the reddened brain; everywhere vessels which during sleep were not visible showed themselves, and arteries and veins could be accurately recognized by their different color. The animal was now fed, and then again sank into quiet sleep; the blood-vessels again became narrower, and the surface of the brain pale as before. The difference in these appearances was the more firmly established as two animals under opposite conditions were observed. Lastly, the state of the vessels was examined with a strongly magnifying lens, and also under weak microscopical power. The trials were several times repeated, with exactly similar results. Dogs were found to be more suitable than rabbits.

“Atmospheric pressure could have exerted no influence in these experiments, for this was the same in both the sleeping and the waking state, and the appearances did not alter when accurately-fitted glass plates were set in the skull-openings. Durham claims that during sleep vascular action and congestion towards the brain exist in a less degree. When awake and when the brain is active, it receives more blood, which moves more rapidly through the vessels, and the brain-substance is more oxidized; for the functional activity of the brain demands a greater consumption of oxygen, and this *vis a fronte* occasions a richer supply of arterial blood, enlargement of the capillaries, and increased change of matter.

“The increased rapidity of the circulation causes a more copious supply of oxygen, and probably also a more active taking up of the products of decomposition into the blood. If fluids are allowed

to stream through a rabbit's intestine which lies in a surrounding fluid, the quicker the stream, the less fluid will transude outward through the wall of the intestine. Now, during sleep the *vis a fronte* diminishes ; on account of their elasticity, the vessels contract more, and the nutritive circulation is predominant ; fewer blood-cells circulate, and the slowness with which blood flows favors the escape of nutritive plasma.

"The immediate cause of temporary suspension of cerebral activity cannot be a failure of active material or its exhaustion by oxidation ; for the already fatigued brain may be brought to renewed activity through suitable stimulation. Durham finds this cause in the products of decomposition, and appeals to the observation that the brain-substance of an animal just killed has a neutral or even a slightly alkaline reaction, but shortly after influence of the atmosphere is, on the contrary, acid. Still, Heynsius, before Durham, found that the quite fresh brain of the sheep or of the ox had an acid rather than an alkaline reaction ; this was confirmed by Funke, who at the same time ascertained that in increased activity of the brain an acid reaction, and in inactivity of the brain an alkaline reaction, is present. Heynsius has further demonstrated that the diffusion or exosmosis of albumen is impeded by acid and is promoted by alkali. Thus, if after long activity, in consequence of oxidation, acid has accumulated in the brain, less albumen transudes out of the blood-vessels ; the change of material, or rather the supply, is less, and only during rest can the acid fluid be taken up and carried away, through which the organ then becomes fitted for renewed activity. This formation of acid would thus be a corrective of over-irritation or immoderate exertion of function. According to Durham, acid prevents the oxidation ; according to Heynsius, it limits the transudation of albumen. Durham thinks that the nutrition is increased during sleep ; Heynsius assumes that there is then increased absorption and diminished deposition of albumen, consequently a weakened nutrition.

"To me it appears more probable that, while during waking the change of matter is increased, with which, of course, a greater supply of oxygen as well as of albumen must be combined ; during sleep, on the contrary, the deposition and nutrition are more active, whereby, at the same time, the acids formed are carried away.

“ We may consider that sleep arises not so much from lessened supply of arterial blood as from diminished change of matter, thus from diminution in the supply and apposition of new substance. Diminished supply and weaker oxidation may also occur if in derangement of the circulation the vessels are considerably distended with blood, and if from powerful resistance the circulation becomes slow ; the renewal of the blood in the capillaries then follows too slowly, and its venosity increases. Coma and sleepiness thus need not be always the consequence of congestion and sluggish circulation ; contraction of the vessels can likewise induce it, as well as a lessening of the oxidation and nutrition through preceding exertion and formation of acid which impedes nutrition. The essential cause in both cases lies in a lessening of oxidation. Thus, also, the fœtus appears to be in a lethargic condition before the commencement of respiration, so long as its blood is only weakly arterial and oxidation is at a low grade ; only after the beginning of respiration does it awaken out of this state, and it gives evidence of this by half-voluntary movements.”

LECTURE II.

GENERAL HYPERÆMIA OF THE BRAIN.

Definition—Active Hyperæmia—**Causes**: Emotions, Fevers, Diseases, Slight Resistance of Capillaries, Pressure, Malaria, Cold, Atrophy of the Brain, Paralysis of Vaso-Motor Nerves, Alcohol, etc.—Irritation of Vaso-Motor Nerves: Poisons, Alcohol, Excessive Mental Work—Passive Hyperæmia—**Causes**: Strangulation, Pressure, Expiratory Efforts, Impediments to the Heart's Action, Compensation, Altered Structure or Function of the Lung—Post-Mortem Changes—Active Congestion—Anæmia produced by Collateral Edema—Forms of Hyperæmia—Symptoms of Mild Hyperæmia—Severe Form—Delirium—Insanity—Hallucination—Illusions—Apoplectic Form—Common Symptoms—Diagnosis—Prognosis—Treatment—Partial Form of Hyperæmia.

GENTLEMEN,—Since the publication of the first edition of this work in 1878 certain authorities have expressed doubts regarding the existence of cerebral congestion, and would seem to claim that the subject is barely, if at all, entitled to nosological existence. Seguin, of New York, for whose writings I entertain the most profound respect, and whose opinions we shall frequently quote, in a recent article upon the "Treatment and Management of Neuroses" states that "the condition known through a feat of imagination as 'hyperæmia of the brain,' which has been quite a prominent figure in our array of diseases during the last twenty years, and which is now beginning to be studied and reclassified into more correct clinical types, was and is still the object of treatment by the free use of bromides. The creation of the 'disease' was mere theorizing, and its treatment dictated by apparently logical deductions from a fanciful premise. We know that many of those cases which provisionally I have for several years designated as 'paræsthesia about the head' (the most common symptoms being fulness, tightness, numbness, emptiness, and some pain in the head, imperfect sleep, nervousness and hysteroid conditions, flushing of the face, with cold extremities, asthenopia, tinnitus aurium, apparent loss of memory, etc.) are really dependent upon eye-strain (especially those in which occipito-vertical

symptoms predominate), lithæmia, dyspepsia, and not rarely upon a weak heart or mitral regurgitation. The time has not come for a successful or final analysis of this symptom-group, but the belief in the original conception of its hyperæmic nature is fast disappearing."

Gowers, in his "Diseases of the Nervous System," also makes the following comments: "Of all regions of cerebral pathology, that of congestion of the brain is perhaps the most obscure. We have very little precise knowledge regarding it, and, as is often the case, theory has flourished in proportion to the deficiency of fact. It was long thought that the state of the vessels of the brain after death corresponds with their condition during life, and the post-mortem distention was accepted as a proof that any preceding cerebral symptoms were due to congestion. The fact was unobserved or ignored that a similar condition of the brain is equally common when there are no cerebral symptoms during life, and depends chiefly on the mode of death. Hence an extensive symptomatology was elaborated and built upon an erroneous foundation, and it has to some extent survived its evidence. Moreover, congestion of the organs seems to afford so satisfactory an explanation of derangement of their functions, that the temptation to assign the condition as the cause of the symptoms has proved irresistible to unscrupulous practitioners. In this way, also, a symptomatology has grown up, and even statistics have been amassed, the value of which may be estimated from the fact that in one modern text-book the history of cerebral congestion has been manifestly written from cases of pure hypochondriasis. On the other hand, partly by a reaction from this extreme, some have doubted even the possibility of the condition. *The truth lies between the two extremes, but its precise position will long be undetermined.*"* Opportunities of ascertaining the exact pathological condition in these cases are very rare, and, if no visible lesion is found, it is not always certain that the symptoms observed during life were the result of congestion. Hence there is room for wide difference of opinion, even among those who strive to keep their minds unbiassed. It is certain, however, that the cases in which symptoms of definite character

* Italics my own.

and considerable degree can be reasonably ascribed to this cause are far from frequent."

Trousseau, commenting on this subject, says, "The existence of cerebral congestion is not contested; but it has been singularly abused, in order to explain cerebral phenomena in the production of which congestion plays no part whatever."

I must say, gentlemen, that I cannot myself understand momentary congestions of the brain, which play so great a part in the diagnosis of many physicians. But this does not impair my faith in the existence of hyperæmia of the brain, nor do the antagonistic views of eminent authorities influence me in this respect. In fact, I believe that the study of cerebral hyperæmia and its collateral phenomena constitutes the keystone in the analysis and explanation of the greater part of the vast domain of cerebral pathology.

Notwithstanding the great advance of neurological knowledge in the last decade, we still claim that the classic views of Niemeyer on this subject have been but little, if at all, improved by recent writers, and we therefore will still adhere, as in our former lectures, to his teachings and classifications of this subject.

I have said that cerebral hyperæmia is justly entitled to nosological recognition. This statement is sustained by Spitzka, as follows: "While this change in our views is the natural result of progress in experimental pharmacology and pathology, it does not justify the extreme assertion that there is no disorder of the brain-functions deserving the name of congestion and hyperæmia. This assertion seems to have been provoked by the careless manner in which these terms have been employed to designate conditions which are in reality the most different in nature that can be well conceived. No one familiar with the extent to which the term 'congestion of the base of the brain' has been abused in this country will marvel that the reaction provoked by it has overstepped the boundaries of cautious criticism. That there are physiological hyperæmias of the brain is now universally admitted; the most recent experimental observations, indeed, conform most closely to the claims of the older investigators. It naturally follows that pathological hyperæmias are both possible and probable, and, even if the observations in the dead-house do not strongly sustain the existence of pathological hyperæmias and

congestions independently of gross disease, clinical analysis and the gratifying results of appropriate treatment justify us in retaining these designations in our nomenclature with the limitation here implied."

We are compelled to admit that some of Spitzka's conclusions are more than probably correct, in view of some of the older theories of the etiology of congestion of the brain, especially, for example, as induced by insolation. Spitzka's conclusions in some respects agree with those of Niemeyer; the theories of older writers are at variance with many pathological observations. Spitzka observes that "Arndt, who had the opportunity of studying over one hundred cases occurring in the course of a forced march of a division of infantry from Berlin to Pankow, many of which terminated fatally, found almost uniformly a pale brain, with peculiar color-changes denoting rather structural than circulatory trouble."

As regards the effects of mental overstrain producing cerebral hyperæmia, Spitzka quotes Nothnagel as one of the more cautious writers who refuse to commit themselves to the view that the result of mental overstrain is a simple cerebral hyperæmia.

In an important foot-note, Spitzka makes the following observations: "It has repeatedly happened during the past decade that young persons competing for admission to higher institutions of learning in New York City through the channel of a competitive examination died with symptoms of cerebral irritation; the death-certificates in several such cases assigned meningitis or cerebral congestion as the cause of death, and attributed the disorder to mental overstrain. It is not so much the intellectual effort that has proved hurtful to the pupils as the emotional excitement attending on all competitive work, the dread of failure, the fear of humiliation, and anxiety developed by the evident futility of the cramming process. Some years ago I recorded the results of some inquiries on this head in the following words: 'The mental-hygiene sensationalists, who periodically enlighten the public through the columns of the press whenever an opportune moment for a crusade against our schools and colleges seems to have arrived, are evidently unaware of the existence of such a disease as *delirium grave*, and ignorant of the fact that the disorder which they attribute to excessive study is in truth due to a generally

vitiating mental and physical state, perhaps inherited from a feeble ancestry. Our school-system is responsible for a good deal of mischief, but not for meningitis.' (Insanity, its Classification, Diagnosis, and Treatment.) Since then I had an opportunity of obtaining an excellent description of such a case which had been attributed to the combined effects of malarial and educational overstrain, presenting opisthotonos, fulminating onset, and an eruption!"

I do not entirely agree with Spitzka when he maintains that "The whole list of causes of what is commonly designated cerebral hyperæmia, congestion, and engorgement may be gone through with and similar modifying statements be found to apply to them. The nearest approach to an ideal cerebral congestion is that found with acute alcoholic intoxication." (See Spitzka upon "Anæmia and Hyperæmia of the Brain and Spinal Cord," Pepper's System of Medicine, 1886.)

In opposition to the usual rule of study in most diseases, it is far more important in cerebral hyperæmia to consider the *etiology* rather than the *semiology*; this is apparent for two reasons: in the first place, as will later be explained, the symptomatology of cerebral hyperæmia is analogous to that of cerebral anæmia; and in the second place, it is only by the study of *extra-cranial* conditions that we can possibly understand and explain the phenomena of cerebral congestion. By *hyperæmia* is meant an undue or excessive determination of blood to a part. Hyperæmia of the brain is generally divided into two kinds, *active* and *passive*. By *active* hyperæmia we mean *arterial* or acute fluxionary congestion. By *passive* hyperæmia we understand *venous* or congestive hyperæmia.

The causes of *hyperæmia* are numerous, and among them we find, as factors of acute fluxionary hyperæmia:

1. Increased cardiac action resulting in increased capillary congestion, the arteries under these circumstances containing more, the veins containing less, blood.
2. An *independent cardiac hypertrophy* is another cause of cerebral congestion, especially when the former state is not compensatory. The latter condition is more particularly found in alcoholism and in persons subjected to excessive muscular exercise.
3. The next cause of acute fluxionary hyperæmia I fully agree

with Niemeyer results from "*too slight resistant power of the afferent blood-vessels*," whether this be congenital or acquired. When the cerebral arteries have delicate, thin walls, so that they yield to an increased pressure of the blood sooner than the other arteries of the body do, and hence, when the action of the heart is only moderately increased, fluxionary hyperæmia of the brain is induced, it is customary to say that the person so affected has a tendency to "rush of blood to the head."

4. The *emotions* and mental excitement are also factors in the etiology of cerebral hyperæmia. We have all experienced the rush of blood to the head, preceded by violent throbbing of the heart, and followed by heat in the upper part of the body, caused by violent mental emotions. Ordinarily these emotions are not of a grave nature, and consequently their effects are transitory. It happens, however, that their violence may be such that the hyperæmia may result in severe—nay, fatal—consequences. Hyperæmia in this case is due in part to increased cardiac action and partly to paralysis of the vaso-motor nerves.

5. Another frequent cause of hyperæmia is *fever*, which produces this morbid condition by increased cardiac action, blood-poisoning, and excessive elevation of temperature.

6. Certain pathological conditions produce hyperæmia, as hypertrophy of the left ventricle of the heart, which is sometimes present in chronic renal disease, and non-complicated and over-compensatory hypertrophy.

7. Undoubtedly one of the most direct causes of hyperæmia is pressure *by tumors upon certain portions of the aorta*. Most of the blood supplying the brain being carried there by the carotid arteries, if in any way a pressure upon the thoracic or abdominal aorta should be exercised, it is evident that the greater volume of blood must be carried upward, and consequently to the brain, thus giving to this organ an undue supply by increased lateral pressure in the carotids.

8. In some forms of *malarial* poisoning we have hyperæmia during the algid stage, produced by diversion of the blood from the peripheral cutaneous vessels to the more deeply situated organs in consequence of spasm of the smaller vessels. It frequently happens that extreme cold, in this manner, becomes a grave cause of hyperæmia. The explanation in this case is ex-

tremely simple. We all know that cold constricts the capillaries, and consequently produces an increased current to the internal parts. A great deal of this blood goes to the brain, and, as it is arterialized, it irritates the nervous system and produces symptoms of congestion. As mentioned by Watson, men perfectly sober have been arrested in the streets during very cold weather for being drunk, when the cause of their strange behavior was traced to the effects of the extreme cold.

9. *Insolation, sudden arrest of hemorrhoidal or menstrual discharges, night vigils, excessive indulgence in the pleasures of the table, and position* (as may be instanced in a tribe of South American Indians who stand upon their heads some time prior to undertaking a long journey, which they assert prevents subsequent fatigue and exhaustion), are all prolific sources of dangerous attacks of active cerebral congestion. Spitzka says, "The suppression of habitual discharges, of the hemorrhoidal flux, and the cessation of menstruation, are associated in many instances with the more formidable grade of cerebral hyperæmia. Many phenomena of so-called climacteric insanity depend on congestive states. The sudden closure of an old ulcer or the removal of hemorrhoids in advanced life has in some well-established instances provoked alarming seizures not unlike those noted with parietic dementia. The chain of proof establishing the direct influence of physiological and pathological discharges on the vascular controlling apparatus of the brain is most complete. Not alone cumulative clinical observation, but the occasional happier result of therapeutical procedures based on this supposed interdependence, supports it. Thus, the congestive cerebral state is recovered from when the menstrual or hemorrhoidal flow is re-established, or an issue is formed in the nape of the neck, or an old ulcer is allowed to reopen."

In corroboration of this, I had occasion to observe many years ago, at the St. Louis City Hospital, the case of a woman who while menstruating had scrubbed a pavement on a cold day with bare feet; the discharge was immediately suppressed, and she died on the fourth day, from the effects of the most intense cerebral hyperæmia that I ever witnessed.

After commenting upon this subject as above quoted, Spitzka, in a highly important foot-note, observes, "The treatment of parietic

dementia, particularly of the congestive type, is also based on this relation. The irritating antimonial ointment and issues in the nape of the neck, etc., have been lauded by older observers, and in two of my own cases have had the best results,—in one, indeed, with established *restitutio ad integrum* of now nearly two years' duration. I am inclined to suppose that its abandonment is due to an improper selection of cases. In the ordinary premature senility and syphilitic types such treatment is altogether ineffective. It is applicable but to a minority of cases at best, and to them only at an early period. It is probably to a similar form of congestion that Bouchut refers when ('*Les Névroses congestives de l'Encéphale*,' *Gazette des Hôpitaux*, 1869) he speaks of a cerebral hyperæmia developing under the mask of a meningitis,—an expression that may be allowed if understood in the same sense as the comparison between hydrocephaloid and hydrocephalus."*

Speaking of the "neural irritability" from cerebral hyperæmia of professional men suffering from worry incident to professional life and irregular living, so provocative of gastric disturbances, with resulting insomnia and constipation, Spitzka makes the following statements: "In such a case the insomnia, usually due to neural irritability, if not aggravated by an existing dyspepsia, leads to such a one, and a *circulus vitiosus* familiar to all physicians is established. Each of the factors concerned involves strain of the cerebral vaso-motor apparatus, but none more so than insomnia. It is not so much the intensity of the strain as its long duration, and the exhaustion of the centre which in sleep is supposed to be at comparative rest. This rest is not obtained, and, in conformity to the laws of neural exhaustion, that centre becomes morbidly irritable. Now, gastric irritation is competent to produce a reflex influence on even the healthy cerebral organ; to do so it must be a severe one; but with the class of persons alluded to the slightest indiscretion in food or drink is sufficient to set up reflex vertigo or headache. The current theory regarding these symptoms is that they are due to stimulation of the vaso-constrictors and ensuing cerebral anæmia; but the subjects before us will usually be found to flush up instead of becoming pale, as in simple vertigo

* Spitzka, article on "Anæmia and Hyperæmia of the Brain and Spinal Cord," in *Pepper's System of Medicine*, vol. v., 1886.

a stomacho læso, or, if there be initial paleness, there is a secondary flush, as if the tired arterial muscle had become exhausted by the effort at obeying the reflex stimulus. In addition, a profuse perspiration sometimes breaks out on the upper part of the body."

We must confess that this distinction of our learned author between the action of the vaso-constrictors in these cases and the mechanism of vertigo a stomacho læso, so graphically described by Trousscau, seems rather too subtle for general appreciation.

10. *Atrophy of the brain* is still another source of hyperæmia. Atrophy of the brain itself may occur in many ways; but when it exists, and a portion of the brain has wasted away, a vacuum forms,—or rather would form, were it not that, in consequence of a compensatory vascular dilatation, the wasted brain is replaced by blood; and thus we have here also another cause of undue determination of blood to that organ.

11. One more cause of hyperæmia, the result of a diversity of influences, is yet to be enumerated. I refer to *paralysis of the vaso-motor nerves*, accomplished in many different ways, prominent among which are: section of certain nerves, emotions, excessive intellectual labor, narcotic poisons, and, very frequently, abuse of alcoholic stimulants. In order fully to understand the manner in which paralysis of the vaso-motor nerves causes hyperæmia, we must first consider the functions of these nerves, the neuro-physiology of the vessels to which they are distributed, and the presiding influence exerted by these nerves over their proper innervation. The blood-vessels are furnished with a certain elastic coat, which, by alternate expansion and contraction, regulates the flow, and consequently the supply, of blood to certain parts. As the stimulus to the organs of the body is derived from nervous centres, and conducted by nerves, it follows that this contractile coat must also be supplied by a nerve, which in this case is derived from the great *sympathetic*, from which all vaso-motor nerves emanate.

The nerves subserving this function are called the *vaso-motor*,—in other words, nerves regulating the movements of the vessels. When no pernicious influence acts upon these nerves, they perform their functions normally, unless the coats of the vessels have undergone some change of structure rendering it impossible for

them to respond to the impulses of the nerve-force. But where injurious impressions are exerted, they seriously interfere with the appropriate action of the nerve, and more or less impede or augment the circulation. When a vaso-motor centre or its nerve is *irritated*, the arterioles thereby supplied immediately *contract*. But when a *paralysis* of these nerves exists, the *reverse* takes place: the nerves lose their excitability and are unable to transmit the command for contraction, the arterioles remain as a consequence dilated, and hyperæmia results.

It has already been observed that the causes of such a paralysis are various, those most prevalent being such poisons as opium and other narcotics. Alcohol is the most common source of mischief in this respect. Excessive intellectual work is also mentioned by some writers as a prolific and disastrous cause of hyperæmia.

Having heretofore considered the different causes of hyperæmia of an active character, it behooves us to review some of the causes of passive, venous, or congestive hyperæmia.

Passive hyperæmia, or *congestive hyperæmia*, it is well to recollect, may be produced by strangulation, pressure on venous trunks, violent expiratory efforts "while the glottis is contracted," impediments to the functions of the heart, and altered or pathological conditions of the lungs.

There is nothing which exercises so much power in producing passive hyperæmia of the brain as strangulation: this is best exemplified in hanging, where congestion immediately takes place. The *modus operandi* is, that it simply opposes or prevents the return of venous blood to the right side of the heart by a mechanical obstacle. Pressure upon venous trunks produces results identical with those of strangulation, by obstructing the return of venous blood to the heart. Tumors in the neck pressing upon the jugular vein, or aneurism in the thorax pressing upon the vena cava descendens, may be cited as causes having a similar result. The violent expiratory efforts previously alluded to are often witnessed in persons playing upon wind-instruments which require the forcible expulsion of air from the lungs. Loud and prolonged singing and speaking, the straining of parturient women and of persons at stool, and violent muscular exercise, may also be classed in the same category of causes. A better

illustration is afforded by the rapid succession of expiratory acts in whooping-cough.

The impediments to the heart's *function* are numerous, and generally productive of hyperæmia of a congestive form. Let us suppose, by way of illustration, that we have a case of disease of the aortic valves, accompanied by regurgitation. How will this produce hyperæmia? The arterial blood has not perfect exit from the left ventricle of the heart during the systolic contraction, and hence prevents the free entrance of the blood, through the mitral valves, returning from the lungs; the natural result is that the arterial blood, being unduly retained in the lungs, obstructs the venous blood flowing from the right ventricle, the obstruction in the right ventricle is soon appreciated by the right auricle, and finally, the free entrance of blood from the vena cava descendens being obstructed, we will have passive congestion of the brain. There is in nature, however, a grand force which exerts itself whenever occasion demands,—the principle of compensation. In obedience to this salutary law, one organ being diseased, another performs double labor, or one part of an organ being injured in any way, the healthy portion makes up for the deficiency by increased action. These compensations will necessitate an hypertrophy, which generally occurs, especially in cardiac lesions of a valvular nature. The left ventricle hypertrophies and performs increased duty, expelling the blood from its cavity with augmented force. When the compensatory action is sufficient, there will be no congestive hyperæmia resulting.

The last cause we have to consider is the alteration in the physiological functions in the lungs, with or without change of structure, resulting in simple capillary obstruction. In hydrothorax there is a liquid effusion, and in emphysema a larger volume of air presses upon the capillaries, obstructs the pulmonary circulation, and impedes the flow of blood from the right ventricle through the branches of the pulmonary artery. The right auricle becomes engorged with blood, a large column of which fills the jugular veins, engendering passive hyperæmia of the brain. It must be observed that in both these cases nature often endeavors to compensate for the lack of power of the right ventricle to propel the blood through the obstructed capillaries, by effecting hypertrophic changes in its walls.

Anatomical Appearances.—It is not always easy to determine, upon *autopsy*, the previous existence of hyperæmia. The amount of blood in the brain after death corresponds to a great extent to the conditions previously existing. The effects of passive hyperæmia are quite different from those of active hyperæmia, and not very liable to be confounded with them. In *active hyperæmia* some of the principal results are great vascularity of the affected parts, numerous *puncta vasculosa*, and the exudation of serum, occasioning œdematous infiltration, especially of the pia and in the cerebral tissue. In *passive hyperæmia* (congestive) we may find the brain hyperæmic, with a venous plethora, at the expense of an arterial anæmia; in *chronic* congestions, *l'état criblé* of Durand-Fardel. However, none of the conditions above described are conclusive of the cause, which may depend upon many different circumstances, and indeed it may be said, with Gowers, that "there is scarcely any pathological anatomy of congestion of the brain. Simple active congestion disappears after death in every organ." Indeed, in post-mortem examinations, incorrect deductions as to the existence of hyperæmia are very apt to be made, the arteries at the base of the brain being naturally large, and oftentimes their post-mortem fulness is undoubtedly influenced by the tendency on the part of the blood to gravitate to more dependent parts. The distended and tortuous blood-vessels on the top of the brain are venous, and quite capacious. *These vessels are almost always found empty after a long-continued and exhausting disease.* The vessels outside of the brain, therefore, afford no definite evidence of hyperæmia, nor does the presence of a large or a small quantity of blood in the substance always give us any positive information on which to build our conclusions for the supposed presence of ante-mortem congestion.

The brain-substance receives blood from capillaries proceeding from the pia mater, and the vessels of the cerebral substance proper are too minute to be seen by the naked eye. We can, it is true, judge of the quantity of blood these capillaries contain, approximately, by making a transverse section of the brain, causing them to become apparent to us as little dots, called the *puncta vasculosa*. But even these are not thoroughly significant, for the rapidity or freedom with which the blood exudes from them is often due to change in its constitution. When the blood

runs slowly, it is often due to a state of hyperinosis; when freely, it is often owing to a deficiency of fibrin in it (*increased fluidity*), which frequently happens in cases of dyscrasia. Here, once more, we have no infallible test for determining the presence or absence of hyperæmia. The knowledge of the effects of hyperæmia will undoubtedly throw much light upon our investigations, and enable us to recognize many pathological conditions which would otherwise escape our observation.

In cases of active congestion, the *capillaries* are distended, the blood moves very slowly through them, and perhaps also, in consequence of some cause exercising a constantly-increasing tension on the coats of the vessels, there is developed upon the part of the capillaries a proneness to transude serum into the perivascular spaces and the pia, and into the cerebral substance; likewise into the ventricles and subarachnoidean spaces in chronic congestions. When the congestion continues for some time, this last result is undoubtedly the danger to be feared.

Serous transudation is known as

COLLATERAL ŒDEMA.

It is evident that the collateral œdema cannot distend the membranes, though it nevertheless exerts a constant pressure. Does it compress the brain? Of course not. The fact of the brain's incompressibility has already been established. What, then, sustains the constraining impulse? It compresses the capillaries, the very vessels whence it *originated*. These vessels being the only elastic portion of the brain, and having the pressure of the collateral œdema exercised upon them, their calibre is diminished or entirely obstructed in some instances, and an anæmia is the result. In other words, the parent vessels are strangulated by their own progeny, the collateral œdema. You must not suppose, however, that collateral œdema necessarily occurs in all cases of congestion. Its amount is proportional to the intensity of the congestion. In death from congestive hyperæmia, therefore, we generally find the affected districts more or less anæmic.

Gowers, referring to passive congestion, says, "It is probable, however, that the spaces around the vessels, which arise by a dilatation of the perivascular sheaths, are increased by mechanical congestion, although here again, apart from congestion, the varia-

tions met with are so great, and the size of these spaces is often so considerable, that the influence of congestion upon them cannot be regarded as proved. Even in young persons their size is often considerable. . . .

"Bulgings of the capillaries have been occasionally seen. After asphyxial modes of death it is common to find that vessels here and there have given way, so that the lymphatic sheath is filled with blood. Blood-pigment in the sheaths has been found in cases of slighter long-continued congestion (Bastian). Lastly, minute microscopic hemorrhages into the cerebral substance may be found almost constantly in these cases. . . .

*"While the pathological anatomy of congestion is thus to a considerable extent negative, it is important to point out that this affords no reason for doubting the occurrence of the condition, since on other parts conspicuous congestion during life may leave no trace."**

The child's brain is more vascular than that of the adult or of the old man.

Some portions of the brain are much more vascular than others : they may be classified as to their vascularity as follows. In the first place, the gray substance contains more vessels than the white ; the corpora striata contain a great number of small vessels in their cortical zone ; the optic thalami are next in order ; then the corpus callosum ; then the cerebellum, which is particularly vascular in its peripheral part about the level of the corpora dentata. Sections of the pons present only a small number of vascular points.

Other facts should be remembered when making post-mortem examinations.

Ante-mortem congestion may be effaced after death. It is necessary to determine in such cases whether the cerebral tissues and the ventricles have undergone any modifications which follow in the train of ordinary congestion.

Localization of results in such cases has its influence, because dorsal decubitus will often show congestion in the occipital region, as the result of a post-mortem and not of an ante-mortem condition.

* Italics my own.

Cadaveric congestions and those associated with the agony of death are limited to the membranes of the brain and its surface, whereas pathological cerebral hyperæmia penetrates into the cerebral substance and ventricles.

Cerebral congestion is not always equally marked or limited over the entire extent of the brain; ordinarily occupying both the white and the gray substance, it may predominate in one more than in the other.

The membranes are never adherent to the cerebral pulp in congestion of the brain.

Retinal congestion during life is often associated with cerebral hyperæmia.

Collateral œdema is accompanied by whiteness of the brain-substance, which is moist and shining.

The development of Pacchionian bodies is not a necessary concomitant of cerebral congestion.

SYMPTOMS.

By many authorities brain-symptoms are divided into symptoms of *irritation* and symptoms of *depression*: the former are due to increased excitability, the latter to diminished excitability of the brain. There is no absolute order of their development: those of irritation usually precede those of depression; sometimes they blend together from the very beginning; at other times, though rarely, the latter are the only ones manifest from the onset.

Niemeyer, who has given the most satisfactory explanation of the philosophy of these conditions, states, with respect to the symptoms of irritation, that "experience shows that nerves passing through bony canals in company with blood-vessels are thrown into a state of increased excitability and morbid excitement by overfilling of these vessels; the nerve-elements of the brain, enclosed by the dura mater and skull, are in a like condition when the cerebral vessels are overfilled."

Brain-pressure, therefore, is of some significance in the explanation of the former condition, but not in that of the symptoms of depression or paralysis.

The latter—namely, the symptoms of depression in *congestive* hyperæmia—are explained by the failure of the venous blood

to escape freely from the brain, whereby the entrance of fresh arterial blood into the capillaries is prevented. The absence of oxygenated blood, therefore, plays a most important part under these circumstances.

"It is for this reason," continues Niemeyer, "that the symptoms of cerebral hyperæmia are very similar to or exactly identical with those of cerebral anæmia; this is true in regard to *congestive* hyperæmia and anæmia, and the explanation of the correspondence is easy. In both cases the brain lacks its new supply of arterial blood."

The symptoms of depression in *acute fluxionary* hyperæmia are even more readily explained by the last-mentioned author, upon the hypothesis that the collateral œdema of the brain results in "capillary anæmia, a condition directly opposite to the original hyperæmia;" in consequence of which, the arterialized oxygenated blood, if present at all, is greatly diminished in quantity.

Before studying the symptomatology proper of cerebral hyperæmia, I would remark that the development of brain-symptoms in *fever* is not the result solely of cerebral congestion, but is to a great extent induced by the increased temperature of the blood and the consequent augmented tissue-metamorphosis and other states of the fluids essentially characteristic of fever.

Therapeutically this is a fact of vital importance, and it is for this reason that Graves, of Dublin, immortalized himself "by feeding and not starving fevers;" appreciating the absurdity of combating delirium and other brain-symptoms in these cases upon the pathological basis of congestion, which does not necessarily exist.

The symptoms of irritation and of depression under varying circumstances and conditions appear in the *sensory*, *motor*, and *psychical* domains, sometimes blending and sometimes predominating in one more than in the other.

Convulsions and delirium are typical symptoms of irritation; paralysis and coma are typical symptoms of depression.

There are three forms of hyperæmia,—the *mild*, the *severe*, and the *apoplectic*. A few symptoms are common to all forms of hyperæmia.

Hammond adds other forms to this classification,—viz., the "*paralytic*, *convulsive*, *soporific*, *maniacal*, and *aphasic*."

SYMPTOMS OF THE MILD FORM.

1. Increased excitability and general hyperæsthesia of the nerves of special sense.
2. Contraction of the pupils.
3. Insomnia, or vivid and frightful dreams.
4. Vomiting (spontaneous).
5. Flushing of the face. This symptom, however, is sometimes absent.

In this connection it should not be forgotten that great pallor of the countenance sometimes accompanies the most dangerous forms of hyperæmia.

6. Constipation, accompanied with a very torpid condition of the bowels.

7. Diffused headache, sometimes very violent.

The headache is at times lancinating and increased by movements, noises, heat, and light, making all intellectual efforts very painful, if not impossible. (Jaccoud.)

8. A constant buzzing in the ears (tinnitus aurium).

9. Dizziness in proportion to the severity of the attacks.

The cerebral form of vertigo is objective in character; surrounding objects move, not the patient himself; closing the eyes relieves it, and does not relieve sympathetic disturbances. (Da Costa.)

When diseases of the posterior cranial fossæ are described, special distinctions as to the character of vertigo will be made. (See termination of lecture on Partial Cerebral Anæmia.)

10. Motor and sensory symptoms of excitation, and later on of depression, more or less marked.

11. Light and noises are badly tolerated.

12. More or less psychical disturbance.

13. Vomiting, which when protracted is frequently accompanied with a slow pulse.

14. Sensations of heat and pulsations in the head, with violent beating of the carotids.

15. The conjunctivæ are often, but not always, injected.

The symptoms of the mild stage of cerebral congestion may frequently return when the causes for their excitation are repeated. They may last from a few hours to several days.

SEVERE FORM OF CEREBRAL CONGESTION.

In the severe form, characterized by great intellectual excitement and disorder, nearly all of the above symptoms present themselves, increased in intensity and accompanied by delirium, hallucinations, illusions, and delusions. Simple delirium is a wandering of the mind, often attended by fever, of which it may be the result. It is not a symptom, necessarily, of hyperæmia, as fever is absent in the latter, but rather points to blood-poisoning or excessive tissue-oxidation, accompanied by increased elevation of temperature.

A man is in a certain sense insane when he is unable to use his mental powers to dispel hallucinations which are nothing but imaginations of things having no real existence outside of the patient's brain; he then labors under a delusion. If he imagines animals not actually present jumping upon his bed, he labors under an hallucination; while if he perverts the impression received in his brain through the senses from external objects having a real material basis of existence,—as, for instance, if he imagines a chair really present to be a living, moving object, approaching or threatening,—he then has illusions.

Delirium tremens should never be confounded with ordinary hyperæmic delirium, from the easily-recognized fact of the involuntary tremor, coinciding with “the characteristic good-natured and loquacious delirium” almost pathognomonic of the former.

In the grave form, sensorial disorders are the first phenomena to manifest themselves, accompanied with illusions and hallucinations. In other more serious forms of this variety of cerebral hyperæmia, delusions and other psychical disturbances are prominent, the latter originating *directly*, and not through sensorial perversions. Muscular agitations and distorted impulses often appear, the patient at times becoming very violent.

If the condition is not soon relieved, all the symptoms become aggravated, the pulse increases in frequency and diminishes in volume, the skin becomes cold and clammy, but the patient has no fever.

With increased muscular agitation and general relaxation, accompanied with involuntary evacuations, the respiration soon becomes stertorous, and stupor and coma close the scene.

Paralysis preceding this condition is rare, and, if present, is generally circumscribed.

In old men the sudden development of delirium at night is the first and only symptom of this form. It is nocturnally recurrent, and is but too frequently followed by a fatal coma.

Durand-Fardel has noticed, as a frequent accompaniment of the senile form of *grave* cerebral hyperæmia, "an abundant sero-mucous secretion of the conjunctiva and of the buccal mucous membrane."

APOPLECTIC FORM.

The apoplectic form is characterized by a sudden abolition of consciousness, sensation, and voluntary motion. The individual suddenly falls, in a condition of perfect relaxation and prostration.

The reflexes are preserved; the insensibility is profound, and involuntary evacuations occur.

Consciousness may be gradually restored after a few hours or days.

Sometimes a temporary paralysis, lasting but a very short time, follows the more purely apoplectic phenomena.

In rare instances a hemiplegia is observed, to explain which nothing can be detected at the autopsy but a *diffused* cerebral congestion. "Some facts show the possibility of a hemiplegic form of paralysis; the autopsy sometimes demonstrates a reason for the existence of this symptom in revealing a congestion predominating upon the side of the brain opposite to the paralysis (Dechambre); but it sometimes happens that the congestion is found equally diffused in both hemispheres, and the most minute examination is incapable of explaining the unilateral seat of the phenomena." (Grisolle).

Jaccoud, commenting upon this subject, remarks, "Taking it for granted that cadaveric investigations in this respect are complete, it is necessary in interpreting these obscure facts to recur to one or the other of the following hypotheses: congestion being admitted as being equally diffused in both hemispheres after death, it certainly was not in the beginning; the sanguineous afflux produced in one portion of the brain an œdematous infiltration more considerable in one part than in the other. Whilst all this is undoubtedly obscure, one fact is certain and deserves to be re-

membered,—namely, a possibility of unilateral symptoms (namely, a paralysis or convulsions) with a generalized cerebral congestion, without hemorrhage.”

The word apoplexy is generally used to express the effects produced by an extravasation of blood into the cerebral substance. By the apoplectic form of hyperæmia, however, is meant that variety the effects of which are somewhat similar, so far as *symptoms* go, to those of apoplectic extravasation. The main symptoms of this form are sudden loss of consciousness, and abolition of sensation and voluntary motion (the *ictus sanguinis* of the old writers).

The different symptoms enumerated as belonging to the several forms of hyperæmia of the brain are also *common to other affections*, and it is essential to be able to discriminate between them, in order not to adopt a treatment based upon an incorrect diagnosis, which in certain instances might prove dangerous, or even fatal; this can be accomplished only by a careful consideration of all extra-cranial causes of hyperæmia. Were we, for instance, to treat a hyperæmic patient for anæmia, we should be likely soon to sign his burial certificate, though the pallor sometimes present in hyperæmia might lead us to think that the patient was suffering from anæmia.

It happens that hyperæmia in children exhibits very severe symptoms which may very closely resemble those of meningitis. The latter disease is very fatal, while the former is not necessarily so: it therefore behooves us to guard against the error of imagining that we have controlled a meningitis—which really never existed. The previous history will throw considerable light upon the case, and greatly aid in the diagnosis. If the child has been very well until the day preceding the attack, if it has suffered from no contusion about the head, or other severe injury, we may after a short lapse of time generally give a favorable prognosis, especially if the convulsions do not recur with frequency and the temperature be normal.

DIAGNOSIS OF CEREBRAL HYPERÆMIA.

The symptoms of apoplexy may be induced by blood-poisoning (uræmia), which we must not confound with the apoplectic form of cerebral congestion. *Insolatio* (sun-stroke), which also destroys

life by suspending the nervous energy, is not the result of hyperæmia, but, it is generally conceded, can be traced to an elevation of the temperature of the whole body so high as to be incompatible with the functions of life, and fatal to the proper performance of the duties of the nerve-centres.

Stomachic vertigo presents symptoms congestive in character, but we should remember that it is never accompanied by loss of consciousness, and the symptoms disappear generally after the action of antidyspeptics to remove the cause.

In congestion the *temperature* is not elevated, which makes it easy to determine whether or not the symptoms be due to fever. All we have to do is to place the thermometer in the axilla, and the diagnosis is rendered more certain.

The symptoms are always of *short duration*; a point of the greatest importance and significance, as a prolongation of the symptomatic indications would cause grave suspicions of serious lesions, or of meningitis.

One marked characteristic feature of the symptoms of cerebral congestion is, that they are *general* and *diffused*, not localized or limited.

In congestion the breathing is regular, not stertorous, and the pulse is but little accelerated, though usually quite strong. In *syncope*, on the other hand, the breathing is impeded, the pulse very feeble and irregular, and the face *remarkably* pale. This last fact must not have undue importance attached to it, since we know that in hyperæmia of the most dangerous type the countenance is sometimes cadaverous.

Loss of consciousness being characteristic of cerebral hemorrhage, epilepsy, and other comatose conditions, we may be unable to determine its cause. If in such a dilemma we wait until the ordinary period for an *epileptic fit* to pass off, and by differential diagnosis exclude other apoplectic states, we can soon discriminate between the presence and the absence of congestion. The phenomena attending apoplectic hyperæmia are transient; in epilepsy they last but a few minutes; and in apoplexy, if the coma be not fatal, they may last for some days.

An epileptic attack is often accompanied by convulsions. Immediately examine the tongue of the patient: you will frequently, though not always, find it lacerated. The control of the sphincter

muscles, in this convulsive disease, is sometimes lost, and you may find an involuntary discharge of feces and urine.

An examination of the abdomen and thorax may lead to the discovery of an *aneurism* or *other tumor* pressing upon some important blood-vessel, or enable us to detect some cardiac or pulmonary lesion causing determination of blood to the head.

SPITZKA'S TABLE OF THE DIFFERENTIAL DIAGNOSIS OF
CEREBRAL HYPERÆMIA AND CEREBRAL ANÆMIA.

| | IN CEREBRAL ANÆMIA. | IN CEREBRAL HYPERÆMIA. |
|---|---|--|
| Pupils. | Usually dilated and mobile. | Usually small or medium. |
| Respiration. | Often interrupted by sighing or by a deep breath, even when at rest. | Normal. |
| Headache. | Either sharp and agonizing, and then in a limited area, or a general dull ache, intensified in the temples and over or behind the eyes. | If localized, accompanied by a subjective and objective (always?) feeling of heat; if general, is compared to a bursting or steady pressure. |
| Activity. | There is lassitude. | There is indisposition to exertion, yet the patient is restless. |
| Temperament. | Lethargic, with exceptions. | Choleric, with exceptions. |
| Intellect. | Inability to exert. | Rather confusion than inability of. |
| Sleep. | Insomnia, interrupted by trance-like conditions, in which the patient is comparatively comfortable. Dreams often pleasant. | Insomnia, with great restlessness, variegated by unpleasant and confused dreams. |
| Upright position of body. | Aggravates all the symptoms. | Either without influence or beneficial. |
| Recumbent position of body and dependent position of head. | Ameliorates. | Aggravates. |
| Influence of acts involving deep inspiration, such as blowing, straining at stool, sneezing, etc. | If any, a sharp headache may ensue, but the other symptoms are not aggravated. | Aggravation. |

As a slight evidence of the facility with which a mistake may be made in the diagnosis and prognosis of cerebral hyperæmia, I will recall a case of more than usual interest, which indelibly im-

pressed upon my mind the necessity for caution in this respect. A very distinguished medical man, about fifty years of age, whose life-long habits of study and excessive intellectual labor had brought on serious brain-symptoms, was placed under my charge. His physicians, who were eminent practitioners, had diagnosed cerebral softening, the result of thrombosis. Upon examination, the patient was found to be perfectly incoherent; his delusions were marked and dangerous in character; his countenance indicated a hebetude amounting almost to imbecility; aphasia was a prominent symptom, and agraphia very pronounced. After desperate efforts to write, he folded with care a paper on which were traced a few illegible hieroglyphics, and, placing it in an envelope with *no address*, handed it to me with a request that I should deliver it to a relative whom he named. Upon my calling his attention to the fact that the name and address had been omitted, he became quite excited and irritated, and insisted that I could not read. Amnesia to a limited extent existed, but it was to me a fact of great significance that, notwithstanding his utter deficiency of normal ideation and the presence of other grave and alarming symptoms, his memory of the past and his interest in the present were far from being greatly impaired. My observation and experience having tended to make me believe that amnesia and decided apathy are the ever-present, concomitant, and characteristic symptoms of cerebral softening, I made a particular note of their partial absence in this case. The patient also presented some sensory and motor disturbances: he was partially hemiplegic on the right side, and the orbicularis oris was implicated, as was evinced in a depression of the right labial commissure, which permitted the saliva to escape and dribble down his face, greatly adding to the stolidity of his appearance. His eyes were injected, and his emotional faculties were preternaturally mobile, as was made apparent by his alternate attacks of weeping and laughing. I was made conversant with the fact that the patient had for several years greatly misused narcotics and stimulants while seeking relief from terrible attacks of neuralgia, to which he had been a victim all his life. This indulgence, as usual, was due to the injudicious advice of various physicians. Medical men are often not a little to blame for the moral, physical, and intellectual wrecks occasioned by their countenancing an imprudent resort in

their patients to these dangerous and potent remedies with a view of relieving temporary pain. They seem to ignore the fire they not infrequently kindle, by the creation of a morbid appetite at times so difficult to restrain, too often, alas ! impossible to appease. The patient had a puffed, bloated appearance, which to an experienced eye strongly indicated the familiar evidences of chronic alcoholism. His mother had died insane, and many members of his family were distinguished for their vagaries and eccentricities, probably being the possessors of the "neurosis spasmodica," living as it were upon the border-land of insanity. The outlook of this case, therefore, was certainly dark ; yet upon a careful investigation and analysis of all the symptoms, and particularly laying stress upon the history of the disease, which was that of alcoholism, I diagnosticated cerebral congestion, the result of vaso-motor paralysis induced by his indiscretions, for which, however, I believe he was in no degree morally culpable. Basing the treatment upon the conclusions I had ventured to adopt, I gave him a preparation containing full doses of Squibb's fluid extract of ergot, digitalis, and muriated tincture of iron, to be taken three or four times daily, regulated his constipation with aloetic laxatives, obviated his insomnia with bromide of potassium, and carefully made him eschew all malt, vinous, and alcoholic stimulants, together with his favorite narcotics.

Had the diagnosis of his previous attendants been correct, such a therapeutic course would have been unjustifiable, not to say destructive. I had the gratification to see him rapidly recover, and in two weeks nearly all his symptoms disappeared, and in two months he was discharged cured. The hyperæmia in his case was doubtless excessive, and the resulting collateral œdema must have been considerable, and a nice point was to ascertain whether the presence of the latter in the delicate brain-substance might not have wrought some disastrous structural changes, in which case the damage would have been irretrievable even after the cessation or disappearance of the primary congestion. Such, however, was not the case, the patient's restoration being complete. It therefore behooves you, gentlemen, to bear in recollection and appreciate the fact that without a proper medical *history* our best efforts at diagnosis will oftentimes be rendered futile.

PROGNOSIS OF HYPERÆMIA.

One attack of hyperæmia predisposes to another, and the repetition of such attacks may result in atrophy, softening, or other serious lesion of the brain, due to the profound nutritive derangement generated by the *dynamic* influences of these repeated fluxions.

A tendency to congestion in other organs, especially the lungs, is a contingency not infrequent, and one which we should anticipate.

The hyperæmia induced by the intemperate use of alcohol is sometimes tenacious, while that caused by anxiety or excessive mental labor, or by the suppression of natural discharges, is generally relieved by the removal of the cause.

TREATMENT.

Whenever it is practicable, the first step in the inauguration of a successful plan of treatment is to ascertain and remove the cause of the congestion. The application of cold to the head is most generally advisable. A very eligible mode of applying it is to introduce pounded ice into bladders or rubber bags and lay them on the patient's head.

Purgatives constitute most efficient therapeutic measures, producing marked derivative effects. Among the many that can be recommended, a combination of jalap and calomel (about ten grains each) deserves special notice.

The local abstraction of blood is often resorted to beneficially in cases of hyperæmia resulting from suppression of certain discharges, especially menstrual or hemorrhoidal; if the former, hot sitz-baths are also useful. The bleeding should generally be done at a distance from the brain, by the application of leeches to the pituitary membrane or the margin of the anus. The abstraction of blood from the general circulation—*i.e.*, by venesection—is undoubtedly the most necessary of all the means of treating certain varieties of congestion of the brain, due to intense collateral hyperæmia, or to increased pressure in the carotids, as a consequence of obstructed escape of blood into the abdominal aorta, and, lastly, in very threatening cases, where it would be dangerous to await the action of milder measures. Venous congestion, with few exceptions, requires bleeding of either a local or a general character.

Hyperæmia from increased cardiac action, uncomplicated by valvular lesions, with undue accumulation of blood in the carotid arteries, imperatively demands such a course. In aneurism producing hyperæmia, or in cases of serious collateral œdema, bleeding should always be resorted to, keeping in mind the indications and urgency of the symptoms. It should not be essayed in cases of valvular diseases of the heart, or paralysis of the vaso-motor nerves, resulting from excessive mental efforts or from narcotic or alcoholic indulgences. These latter require energetic treatment without bleeding, and, in addition to the methods already given, we may resort to warm sinapisms. This last method may often be advantageously employed, the irritating effects produced by rubefacients and vesicants causing a derivative current of blood to the skin and other parts. *Ubi irritatio, ibi fluxus*. Therefore the vesicants and irritants are most advantageously applied to the lower extremities.

We should not overlook the important fact that delirium is increased by temperature. The reduction of the temperature is often effectually accomplished by the judicious use of quinine and alcohol. This has reference, however, to *delirium produced by fever*, and not to that of congestion, as these remedies would, in the latter complication, prove most pernicious. There being no *elevation of temperature*, you have simply to deal with the *mechanical* effects of an undue determination of blood to the parts.

Ergot, the bromides, and digitalis are the most effectual internal remedies in the treatment of cerebral hyperæmia.

Spitzka says that "*ergot of rye*, with its preparations, may be regarded as the cardinal drug in cerebral hyperæmia. There are few drugs in the domain of neurological therapeutics which are so directly antithetical to the pathological state as this one. There is scarcely a case of cerebral hyperæmia that is brought to the physician's attention but may be regarded as being in part due to an over-distention of the cerebral vascular tubes. This is directly overcome by ergot, and the quantity which such patients will sometimes bear without showing signs of ergotism is something remarkable, in notable contrast with the subjects of cerebral anæmia, who are usually very sensitive to it."

The thermo-cautery is often useful, applied to the nucha or over painful spots.

PARTIAL HYPERÆMIA.

Partial hyperæmia is circumscribed and usually limited to *focal* regions.

In other words, it is an affection which disturbs the circulation over distinctly-circumscribed regions. Niemeyer states that, "if an artery or a great number of capillaries be compressed, or otherwise closed, there is fluxion in the collateral branches; if, on the other hand, a vein be contracted or closed, there is a congestion in the capillaries supplying it. Of course there will usually be fluxion at one place, congestion at another, and anæmia at still others, at the same time."

Of course all lesions in local or circumscribed portions of the brain or of one particular hemisphere, such as tumors, clots, and foci of softening, will lead to partial hyperæmia.

The symptoms of the latter are necessarily more localized than those of general diffused cerebral hyperæmia.

Hence we have as local symptoms the so-called "Herd-symptom" (Griesinger). Among these are "circumscribed headache, glimmering or sparks before one eye, or blindness of one eye, contraction or dilatation of one pupil, noise or deafness in one ear, neuralgia or anæsthesia limited to one nerve, but especially spasms, contractions, or paralysis affecting only one half of the body, one extremity, or a single group of muscles, and, lastly, partial disturbance of the mind."

The anatomical appearances are just as difficult to discern as those of general cerebral hyperæmia.

It is well to remember that, while partial hyperæmia produces certain symptoms directly, it is, nevertheless, a fact that associated symptoms exist, superinduced by the primary hyperæmia and the disturbances of the circulation consequent thereupon.

It should, moreover, be remembered that paralyzes which appear and disappear, and other symptom mutations, can be explained only by the appearance and disappearance of collateral cedema and the circulatory disturbances in the vicinity of tumors, abscesses, clots, softenings, and inflammatory foci.

In speaking of excessive hyperæmia and its frequent sequel, anæmia, mention has been made of *collateral hyperæmia* often following this or any other form of anæmia (*vide* prognosis of

hyperæmia). It may be that the correct interpretation of this expression has not been seized, and hence an elucidation of what is meant becomes necessary. This collateral hyperæmia is nothing more than a diversion of the blood, the simple result of some other pathological condition. Supposing the quantity of blood to remain the same in the brain when in a strictly physiological condition, it is evident, on the other hand, that, if by pressure during certain morbid processes the blood is forced out of one part, there will be an undue accumulation of it in another. This is collateral hyperæmia.

We have already seen how hyperæmia produces collateral œdema and resulting anæmia. It will now be readily perceived that anæmia may, in turn, produce collateral hyperæmia of other or adjacent parts, which again, according to its intensity, may terminate in collateral œdema, and often as a consequence of this œdema we have a secondary anæmia. Hence, whenever there is an anæmia in one part of the brain, the patient is in danger of collateral hyperæmia of another portion. Collateral hyperæmia might therefore be legitimately added to the incidental causes of local anæmia of the brain.

Partial hyperæmia of the brain is to be treated by the same principles that are applicable to general diffused cerebral hyperæmia.

LECTURE III.

PARTIAL ANÆMIA OF THE BRAIN.

Definition—Closure of Vessels—Collateral Œdema—Pressure upon Capillaries—Thrombosis—Embolism—Rheumatism—Thrombosis as a Cause of Embolism—Aneurism as a Cause—Artificial Production of Embolism—Effects of Closure—Collateral Circulation—Ligation of Carotid in Man; in Animals—Embolism in Left Side—Why the *Right* Side is generally paralyzed—Fissure of Sylvius—Brain not Gangrenous—Cause of Absence of Gangrene—Cause of Presence of Gangrene—Collateral Hyperæmia—Secondary Anæmia—Compression of Capillaries—Change of Color—Hemorrhagic Infarction—Size of Softened Parts—Anatomical Condition in Anæmia—Pathological Effects of Pressure—Symptoms of Softening of the Brain—Degrees of Functional Derangement—Symptoms of Excitation and of Depression: Amnesia, Agraphia, Aphasia, Hemiplegia—Peripheral Arteries—Variation of Symptoms—Differentiation between Embolism and Cerebral Hemorrhage—Differentiation between Thrombosis and Embolism—Symptoms of Anæmia from Collateral Œdema—Obscure Diseases explained by Collateral Œdema—Explanation of Phenomena of Clot—Symptoms of Pressure by Abscesses, Tumors, etc.—Obscurity of Diagnosis in Brain-Diseases—Views of Charcot, Cohnheim, Heubner, and Duret upon the Terminal Cerebral Arteries—Charcot's Recent Teachings upon the Pathological Anatomy of Cerebral Softening.

GENTLEMEN,—Anæmia, as the term implies, is a deficiency of blood, as regards *quantity*. It is, therefore, the opposite pathological condition to hyperæmia. Partial anæmia of the brain, according to Niemeyer's classification, is dependent upon several causes, that can be placed under three heads, as follows :

1. *Closure of the afferent blood-vessels.* The anæmia from this cause may be general or partial: it is general when the entire encephalon is involved, and partial when limited to one hemisphere, or to a portion thereof. In the majority of cases we have only a *partial* anæmia.

2. *Collateral œdema.* This is a transudation of the serum of the blood into the surrounding tissues. This serum presses upon the capillaries, and is the general result of active or passive hyperæmia. We have already noted its effects while reviewing hyperæmia, and it is evident that, in this connection, we might name hyperæmia as one of the indirect causes of anæmia.

3. *Pressure upon the capillaries by tumors, abscesses, clots of blood, etc.* In all instances we have softening of the brain as a direct result of a continued exclusion of blood therefrom. Let us consider each of these causes. First we have closure of the afferent blood-vessels; this is produced principally by two pathological conditions,—thrombosis and embolism.

It happens in certain affections that the blood is not properly propelled through the vessels. This may occur in various ways. The arteries are all supplied with a fibro-muscular coat, controlled by the vaso-motor nerve accompanying the vessel. This insures the contraction and relaxation of the artery, actions both compensatory and essential to the proper maintenance of the circulation. Arterial elasticity and contractility may be lessened by the pressure of tumors upon the artery, or more particularly by disease of the vessels themselves, as, for instance, calcareous degeneration, or inflammatory action, which gives rise to a disease termed *endo-arteritis deformans*, especially liable to attack aged people. Now, a result of this deficient propulsion of the blood is a *retardation* of the circulation, the inner surface of the arterial trunks being roughened, a deposit of fibrin sooner or later occurring. This process is a slow one, and takes place also in the smaller arterics, greatly impeding, if not preventing, the re-establishment of the collateral circulation. For it must be borne in mind that in *endo-arteritis deformans* the arterioles are in precisely the same condition, pathologically, as the trunks, and, in consequence of their non-dilatability, the collateral circulation is not effected. The clot or fibrinous deposit above described is called a thrombus, and always occurs *in situ*; that is, we find it where the pathological conditions above described are most active. In inflammatory affections the blood is hyperinotic, and consequently will more readily deposit its fibrin, and so in arteritis we find a concurrence of conditions very favorable for the production of thrombosis.

The effects of thrombosis do not materially differ from those of embolism. We have already seen that a thrombotic clot obstructs the circulation at the place of its formation. When a clot obstructs a vessel at a point distant from the place of its formation, it is called an embolus. The latter condition is characterized by suddenness of invasion, and the vessels whose

walls confine or arrest it in its course may themselves be in an entirely normal condition. An embolus may be liberated from different organs of the body, the lungs, heart, etc., and it may be as varied in composition as are its points of departure. An embolus is generally conveyed from the heart, and is usually fibrinous.

Different abnormal states of the system predispose to conditions of the cardiac valves and orifices which may subsequently induce embolism. Rheumatism is a very common cause. In this affection, due to a peculiar *materies morbi*, there is an inflammation of the several fibrous tissues of the body, and there is frequently developed a pericarditis, endocarditis, or thickening of the auriculo-ventricular, aortic, and pulmonary valves. A deposit of fibrin follows, with the formation of concretions and vegetations, parts of which may be detached by the current of the blood, carried away from the heart, and produce embolism in some distant artery. Or, if the original source of the embolus is very friable, it may, as Virchow has shown, break up into minute fragments and constitute capillary emboli.

Thrombosis may sometimes be the cause of embolism, when, for instance, the thrombus is formed in a vein as a result of phlebitis, and a portion of its substance, becoming detached, is carried away in the venous current. When, on the contrary, it occurs in an artery, it closes the artery, and cannot be conveyed along by the current of blood, on account of the diameter of the vessel gradually diminishing. But the venous blood is conducted by vessels whose diameters *increase* in the direction of the current. Therefore, where a thrombus forms, as in phlebitis occurring in certain puerperal and other conditions (coagulation in the uterine sinuses, etc.), the clot, or a portion of it, becomes detached, and is carried along to the right auricle, right ventricle, pulmonary artery, and finally lodges, according to its size, in one of the larger or smaller pulmonary vessels. It is, therefore, evident that when a thrombus in a vein results in embolism, the embolus will always be arrested in the lungs or in the trunk of the pulmonary artery or some of its branches.

As the main cause of cerebral thrombosis (*endo-arteritis deformans*) affects only persons who have passed the meridian of life, it materially differs from embolism in this respect, that the latter

is not confined to any age, and may occur at any moment and under varying conditions.

Embolism, as has been previously stated, may be produced by a diversity of causes, though it generally originates in the heart. It has been known to result from the handling of an aneurism containing a clot of fibrin, whereby the clot or a portion of it became detached, and the patient perished. Physiological experiments have been made with the view of producing embolism artificially, and this has been done by the injection of solid foreign bodies, such as millet-seeds, etc., into the blood-vessels of animals, producing a closure of the afferent vessels, and resulting in disturbances of the circulation in the organs affected.

I will next consider some of the effects of closure of the vessels supplying the cerebrum with blood. Suppose the left middle cerebral artery to be suddenly plugged,—and a very important one it is, supplying a part of the hemisphere with blood. The first result will be an anæmia of that portion of the hemisphere which cannot receive its blood-supply from the artery, now no longer pervious, and, if it be not relieved, that hemisphere will cease to perform its functions, and, the condition persisting, there will be a lack of nutrition, and, as an inevitable consequence, softening.

We have said, when speaking of thrombosis, that in this morbid state the arterioles are in the same condition as the trunks, having lost their resiliency, which is so eminently necessary for the normal propulsion of the blood. It therefore follows that the collateral circulation cannot be established. How is it in embolism, where no pathological state of the collateral vessels exists? Farther on it will be seen that it depends entirely upon the situation of the artery which lodges the embolus whether or not the collateral circulation will be developed, and also whether the artery be a terminal one or not. If the embolus be lodged in an artery *below* the circle of Willis, the collateral circulation will quite possibly occur; but such will not be the case if the plug or embolus be in an artery *above* the circle of Willis, and the portions of the brain deprived of their blood-supply will become anæmic, because the other vessels will not be adequate to send a sufficiency of blood to supply the territory deprived of its nutrient fluid. Where rheumatism, for

instance, is the predisposing cause, there is first an embolus producing anæmia, and this condition will eventuate in softening, and lastly hemiplegia, with more or less abolition of the psychical and motor functions, more or less pronounced, according to the site of the pathological lesion. Whenever, therefore, the embolus lodges *above* the circle of Willis, there is no probability of recovery from the disease, though life may be prolonged by compensatory action of the opposite cerebral hemisphere. It is, for this reason, plainly a much more favorable condition for the clot to lodge *below* the circle of Willis; but even then the situation is sometimes grave, for in man there are difficulties opposing, or at least unfavorable to, the establishment of the collateral circulation, and softening very often results. In corroboration of this statement may be cited the well-known fact of softening sometimes ensuing upon ligation of one of the carotids. This should not deter the surgeon from ligating either of these arteries where necessity demands it. In certain animals the tendency always seems to be in favor of the re-establishment of the circulation, and experiments have been made upon rabbits, where no bad effects ensued from the ligation of three of the four supplying arteries. Of course, where the softening occurs on the left side of the brain, the hemiplegia will be upon the right side of the body; and you will find it to be a well-known clinical fact that the right side is generally the one paralyzed. The reason of this is simple. The right common carotid artery arises from the *arteria innominata*, while the left arises directly from the arch of the aorta, and is, therefore, more in the line of direction of the arterial current; hence it follows that an embolus from the heart will more readily enter the left than the right carotid. The embolus is, therefore, frequently arrested in the left middle cerebral artery, in the fissure of Sylvius. Upon autopsy, it is always prudent to examine this artery, especially when other anatomical explorations have offered only negative results.

Another point which it is well to refer to is that the softened brain does not become gangrenous, although Niemeyer speaks very truly of the condition present being one of necrosis. It softens, liquefies, becomes disorganized, but it does not putrefy or become fetid. The different influences (of atmosphere, temperature, etc.) which are requisite for such a condition are almost en-

tirely excluded, the brain being closely confined and hermetically protected by the skull. Nevertheless, you may find offensive abscesses or putrid collections within the skull; but this happens only when the embolus originated in a necrosed point—namely, an *infectious* embolus—and carried the infection with it from the source whence it emanated. We may have such a condition of things in certain pulmonary affections, as in tuberculosis, where there is considerable tissue-metamorphosis occurring, and a minute portion of gangrenous lung is detached, carried through the pulmonary veins to the left auricle and ventricle, whence it goes to the brain, or possibly to some other part of the body. But, wherever it goes, it carries the putrefactive ferment with it, which will be communicated to any part in which it may chance to be lodged.

As we have already spoken of hyperæmia and collateral œdema as the second cause of anæmia (*vide* post-mortem in hyperæmia, also causes of anæmia), very little remains to be added in this connection; but there is still one point necessary to be dwelt upon. In speaking of excessive hyperæmia and its sequel anæmia in a preceding lecture, mention has been made of *collateral hyperæmia* often following this or any other form of anæmia (*vide* prognosis of hyperæmia). It may be that the correct interpretation of this expression has not been seized, and hence an elucidation of what is meant becomes necessary. This collateral hyperæmia is nothing more than a diversion of the blood, the simple result of some other pathological condition. Supposing the quantity of blood to remain the same in the brain when in a strictly physiological condition, it is evident, on the other hand, that if by pressure during certain morbid processes the blood is forced out of one part, there will be an undue accumulation of it in another. This is collateral hyperæmia.

Anything producing undue *pressure* within the *cranial space* will cause anæmia of the brain. The particular manner in which the capillary compression and subsequent anæmia are brought about will hereafter be considered. For the present it is sufficient to concede the possibility of a partial pressure on the brain. The question naturally arises, "How is it possible to have a partial or limited pressure? Pressure being continuous from molecule to molecule, it will not be localized." This conclusion is erro-

neous, having been arrived at by fallacious reasoning. It was supposed, as Niemeyer states, that the condition was analogous to that of a bottle filled with liquid in which a cork is being forcibly driven. The bottle will break at its weakest point, and not at the point where the pressure is directly applied. There is then a transmission of pressure, continuous and without intermission, and this pressure is applied to the whole bottle, and not localized. In this conclusion, as the last-mentioned author adds, a great fact was overlooked,—viz., the anatomical division of the brain by the *falx cerebri*. The cerebrum is essentially a dual organ, and is thus equally divided into hemispheres; and by still another membrane, the *tentorium cerebelli*, the encephalon is subdivided into three portions.

Now, it is evident that if pressure of a certain degree (pathological or traumatic) be applied to one of these distinct portions of the brain, the membranes above mentioned will effect a limitation of it to the hemisphere to which it has been originally applied, the other hemisphere remaining substantially unaffected. This same law applies to the cerebellum, which may be diseased or suffer from hemorrhage without the cerebrum being perniciously influenced, though not invariably so; as it may happen that the pressure applied may be very great and thus extend beyond a certain limit, when, of course, the membranes would yield. This occurs very seldom. It is clear that sometimes a circumscribed pressure exists in the brain, in no degree transmitted to distant parts; and in this case you will understand that a tumor, for instance, can exercise pressure, and produce regional symptoms due to a partial anæmia.

In what manner does capillary compression (from abscesses, etc.) produce anæmia of the brain? In the simplest way possible. Imagine a sponge filled with water and resting in the palm of the hand. The moment the fingers are flexed upon it, the sponge is compressed and the water exudes. In this manner will a clot, abscess, etc., pressing upon the capillaries, force the blood from them, and produce anæmia in the immediate vicinity.

THE PATHOLOGICAL ANATOMY OF CEREBRAL ANÆMIA.

When speaking of the post-mortem appearances of the brain in hyperæmia, it was noticed that the result of our examination

was often unsatisfactory and deceptive, and that while looking for a hyperæmic brain we might find one which was anæmic. These apparent anomalies and difficulties again meet us in anæmia. During life the anæmic condition of certain parts is readily recognized. For instance, in the face we note the skin, which, owing to its rich vascularity, is very prone to show an anæmic condition, even while the vital processes are still active. But after death we no longer expect to see the healthy, rosy hue, but rather a pale, leaden, cadaverous look, and from this we draw absolutely no information in regard to excessive or diminished vascularity during life. After death, hyperæmic parts often look anæmic, and anæmic parts appear hyperæmic, from the gravitation of the blood and its settling after the cessation of circulation: thus there are many causes which are apt to lead us into error. But we do know that, as a consequence of persistent anæmia, softening ensues, and that, if the anæmia be intense enough, even liquefaction of the anæmic parts may result. Therefore, softening is the condition we have to look for as corroborative evidence of persistent and complete anæmia, and it is easily determined by pouring a gentle stream of water on the brain, when the softened parts will appear more or less disintegrated, according to their different conditions, and be washed away.

It happens that the brain, in various forms of softening, presents either a whitish, yellowish, or reddish appearance; but there is nothing in this to claim our particular attention. There is another condition not to be overlooked,—viz., *hemorrhagic infarction*. Here the brain is dotted with a bloody tint, from minute capillary extravasations; but this is caused by embolism of one or more arterial trunks. In embolism there is always this tendency to capillary rupture and hemorrhage. This occurs in the lung or in other organs just as readily as in the brain; in fact, wherever the embolus happens to be arrested. This is mentioned only to warn the student from mistaking these minute extravasations for the appearances of hyperæmia when, in reality, anæmia exists.

The extent of the softened parts is exceedingly variable, being from the size of a pea to that of a hen's egg, or even larger. It sometimes happens that large portions of the brain are softened without any grave symptoms being manifested during life. This depends entirely upon the importance of the parts implicated; and

although softening of the medullary fibres of the cerebrum might not be attended with many or any symptoms, it is evident that in other parts, as the medulla oblongata or the cortical portions of the brain, softening would give rise to serious and unmistakable symptoms. In some cases of brain-disease, whether the patient dies from an acute or from a chronic affection, we may often be sorely puzzled as to the diagnosis, and post-mortem examination may show nothing satisfactory to explain the symptoms which were present during life. In such cases it is always well to examine the left middle cerebral artery, and we will frequently find it obstructed, more particularly so when sudden paralysis occurred during life. (See Embolism and Thrombosis.)

The *anatomical* condition in anæmia and collateral œdema is very important. The post-mortem examination will reveal, in some instances, a pearly, glistening lustre in the anæmic parts, and the scalpel will be wet or moist with serum.

Among the ordinary pathological manifestations of great pressure is a marked depression of the sulci; the brain, being full of serum, tends to swell, and, as compared with a healthy brain, is remarkably white. Or we may have a protrusion of the *tentorium cerebelli*, or of the *falx cerebri*, which may dip to one side from pressure on the other, or the pressure existing in the lower part of one hemisphere may cause a dipping of the *tentorium cerebelli*. In both instances it will depend upon the amount and cause of pressure, and in mild forms of disease may escape observation altogether.

SYMPTOMS OF SOFTENING OF THE BRAIN.

The causes of partial anæmia, followed by partial softening, are all that we have thus far analyzed. General anæmia will be investigated at a future time.

In considering the symptoms of softening, two important facts must be borne in mind: 1st, there are no pathognomonic symptoms of softening; 2d, the symptoms of anæmia and of hyperæmia are generally very similar. Consequently, a reliance upon any one symptomatic indication will, in most cases, lead us astray. Inasmuch as the treatment in certain cases is of the highest importance, it is necessary to make a correct differential diagnosis to arrive at proper conclusions. This diagnosis can be made only by

patient, deliberate, and careful investigation of the history of the case, and by becoming conversant with all the pathological and etiological facts and data obtainable. We have seen that, although this knowledge is very needful, the student must be careful not to be too exclusively guided or biassed by any single deduction: whilst mindful of the pathological laws of softening, he must carefully weigh and consider the history and probable cause of the disease. With Niemeyer, I would urge you to have due respect for the sequence of events and symptoms in a given case. By the term sequence is meant the manner in which the events or symptoms succeed each other. This will often enable you to draw pretty correct inferences as to the probable cause of the disease. If the phenomena are characterized by suddenness, you may infer embolism; if, on the other hand, the symptoms have been slow in development, you may presume it to be thrombosis, provided youth does not exclude the conclusion. To obviate every source of error, I will successively take up all the forms and corresponding symptoms of anæmia.

Syphilitic disease of the arteries frequently leads to softening, as we will explain more definitely in another lecture. (See lecture on "Syphilitic Nervous Affections," vol. ii.)

The following law must first be established: whenever there is a sudden shutting off of a considerable supply of arterial blood from a part of the brain, there will be an interruption in the functions of that part which will be in direct proportion to the amount of blood excluded. If the blood-supply has only been diminished, a partial or limited suspension of the functions of the part will be the result; but if the supply is completely shut off from an important artery, there will be an entire suspension, in fact, an abolition, of the physiological actions involved.

In anæmia of the brain we find two sets of symptoms: those of *excitation* and those of *depression*. These symptoms may exist separately, or in combination, or may follow each other in alternation. This last is the case where the blood-supply has not been entirely suspended. As the disease advances and the calibre of the artery grows smaller, the symptoms of excitation diminish, while those of depression increase. Where the symptoms of depression alone exist, and persist, we may infer a complete shutting off of the arterial supply.

The symptoms of excitation are : increased or preternatural excitability of the motor, sensory, or psychical functions, headache, hyperæsthesia of the auditory nerve and of the nerve of vision, with consequent photophobia, pain and symptoms of irritation in different parts of the brain, and an implication of all the nerves of special sense. The patient may feel unusually active and quick of perception, and be very bright and uncommonly cheerful. This may be followed by a feeling of lassitude and inactivity, the state of depression. In this state (of depression) there is generally—1st, an impairment of the mental faculties : one of the most easily recognizable symptoms of this is *loss of memory* ; 2d, there is a stolid condition of the individual, a hebetude, accompanied by an appearance of languor and disinclination to exertion, eventuating in mental imbecility ; 3d, the judgment becomes impaired, and all the mental faculties will gradually be noticed to have undergone a retrograde change, finally becoming entirely lost or annihilated. These different affections are generally *progressive*, and we will soon observe, more or less completely developed, amnesia (loss of memory), complicated with agraphia and aphasia.

In agraphia there is an inability to write. This condition does not depend upon any muscular impediment, but is simply the effect of amnesia : the patient has forgotten the alphabetical characters or the art of combining these into words. Aphasia is a partial or complete inability to remember words, the symbols of thought, or to converse. Here there is no difficulty of articulation, but there is a want of ability to recollect the words which are required to express an idea. When certain cortical cells are entirely destroyed, it is impossible to reproduce the memory of ideas.

And so with the affections of the motor system. If the attack be sudden, motion is usually at once destroyed. If it be gradual, we have, first, a limited paralysis, affecting only a few muscles in the beginning ; then the paralysis extends and attacks another part, and so on until gradually an entire and permanent hemiplegia finally results. I say *hemiplegia*, because, as yet, we are dealing with *partial* anæmia.

Now, what are the symptoms peculiar to anæmia the result of thrombosis, embolism, collateral œdema, or pressure upon the capillaries?

THROMBOSIS.

Thrombosis, unless relieved, ends in softening of the brain, which is the result we are to anticipate. How are we to recognize this disease, either in the recent or the advanced stage? Though it is sometimes difficult to make the diagnosis, it is in most cases possible to do so; and although you may not be absolutely certain, you will nearly always recognize the leading features of the affection. Of course you have to keep well in mind everything connected with the development of the case, and be very careful in analyzing the ascertained facts.

Thrombosis, a result of a diseased condition of the arteries (*endo-arteritis deformans*), usually occurs in persons quite advanced in years. Hence *age* is significant, and must not be lost sight of in your examinations. You would hardly think of diagnosing thrombosis in a young, healthy, vigorous man; but where the patient has passed the meridian of life, such a diagnosis would have just grounds to rest upon. But, as age alone is not a sufficient fact upon which to base your diagnosis, you must have recourse to other considerations.

Great importance has been attached by some authors to the condition of the peripheral arteries as a diagnostic sign of thrombosis. Of course it is probable that if a diseased condition of the arteries exist in the brain it may also exist from the same cause in other portions of the body. This may be the case *at times*, but it is by no means so invariably. We may have *endo-arteritis deformans* in the cranium and yet not have it in any other portion of the body, or, *vice versa*, it may exist in some other organ and still be absent in the brain. So that the evidence furnished by the diseased condition of the peripheral arteries may sometimes be *confirmatory*, but nothing more.

The condition of atheromatous degeneration of the arteries in any accessible part of the body is not very difficult to recognize. We generally have in affected vessels a characteristic atheromatous, retrograde, inflammatory condition,—the artery being preternaturally tortuous, rigid, and unyielding, without resiliency, and offering to the touch of the experienced observer an inelasticity whose peculiarity he will immediately recognize; though if the patient has exhibited *chronic* brain-symptoms in connection with a certain

advanced age, and superadded to this we have the condition of the peripheral arteries just referred to, there is a strong presumption of thrombosis.

Still, this presumption is not a certainty, and we need one link more in the chain of pathological evidence to make our diagnosis sure. This will be furnished by a study of the positive and rational symptoms, which are those of irritation and of depression, and these symptoms will alternate or vary in obstinacy or intensity. Paralysis comes on, disappears, or returns; a patient will be very bright one day, and correspondingly dull the following day. This variation has been *incorrectly* considered to be pathognomonic of *softening*. It may be characteristic of anæmia, but certainly is not so of softening; which latter condition, being a result of profound anæmia, is persistent, admitting of no possible variation of symptoms. In softening, the return of the physiological functions is utterly impossible; they are *entirely destroyed*.

This variation or *mutability* of symptoms, especially in paralysis, indicates *only a disturbance of the circulation in the brain*, and simply points to alternations in the extent of the anæmia. But when once a part of the brain is perfectly and permanently anæmic, it softens; and when there is *no* variation in the symptoms present, it points to total abolition of those functions which, for their proper performance, depend upon the integrity of the part of the brain presiding over their evolution. The disturbances of the circulation are due to the following facts: 1st, the gradual narrowing of the calibre of the arteries; 2d, the alternations of anæmia and collateral hyperæmia, the accession of which produces disordered vascular action and corresponding symptoms. But in thrombosis the resulting anæmia is more slowly produced, as are also the changes in structure, causing a *gradual* development of symptoms, the manifestations of definite, progressive pathological lesions.

To recapitulate, we find in thrombosis:

1st. The state of advanced age. 2d. The gradation and duration of the symptoms. 3d. The variations of the symptoms. 4th. The morbid condition of the peripheral arteries; though this is not necessarily present.

EMBOLISM.

Embolism is generally easily recognized, its symptoms being quite plain, and its consequences obvious.

When a man evinces symptoms of softening of the brain, embolism suggests itself. Without a characteristic *suddenness* in the appearance of the symptoms, there is no embolism. In order to obtain confirmatory evidence, inquire at once into the history of the case; and should you ascertain that your patient has been subject to frequent rheumatic attacks, you will examine the heart, and, detecting a *bruit* (diastolic or systolic), may confidently diagnose embolism. Do not fail, therefore, to examine the heart and lungs, and should you find, in addition to the symptoms already enumerated, these organs in a condition favorable for the production of an embolus, you may with safety arrive at the above conclusion.

I have already told you that embolism occurs mainly with strikingly marked characteristic symptoms, and that there is, therefore, less liability to be mistaken in these cases than in those of thrombosis; indeed, the suddenness of the attack alone enables us to diagnosticate with no little precision.

In former lectures I have investigated the different causes producing embolism, having already stated that it is sometimes, though more rarely, a result of thrombosis (caused by a phlebitis or the puerperal condition), under which circumstances the embolus will be found in the pulmonary artery; and I need hardly add that instant death will certainly result should its entire calibre be obstructed.

It sometimes happens that embolism takes place in the spleen, or in the liver, conditions which are of course extremely difficult to recognize during the life of the patient.

"An embolus is a plug of some material which is transported by the blood-current from one situation to another. An embolus may consist of any substance which makes its way into the current of blood. The majority of emboli are fibrinous, being derived from thrombi. But emboli may consist also of portions of a tumor growing within a vessel, of fragments of diseased cardiac valves, of animal and vegetable parasites, of concretions of lime, of pigment, of fat, or of bubbles of air.

"It is plain that a migratory plug or an embolus can hardly be arrested in its course through the veins (with the exception of the vena portæ), since the course of the blood-current in them is from smaller to larger vessels. Embolism, therefore, relates to the arteries, with the single exception of the vena portæ, while a thrombus may be formed anywhere in the vascular tract. A parietal thrombus may be washed off partially or completely by the blood flowing over it. The thrombi, or so-called vegetations, formed on the cardiac valves in endocarditis are a fruitful source of emboli. An end of a thrombus may be broken off by the force of the blood-current from a vessel over the mouth of which the thrombus projects. Finally, fragments may be detached from thrombi in consequence of softening.

"Emboli consisting of oil-globules enter the circulation most frequently after fracture of the bones, particularly when accompanied by extensive laceration of the marrow. Fat set free from marrow-cells enters the open mouths of ruptured veins. The oil-drops collect chiefly in the pulmonary capillaries, where they can be recognized only with the microscope. They may pass through these capillaries and lodge in the glomeruli of the kidneys, in the cerebral capillaries, and in other capillaries of the body. In most cases emboli of fat are innocuous. They may, however, occlude a sufficient number of pulmonary capillaries to occasion severe symptoms, or even death, especially when the patient is already much prostrated. An excretion of fat by the kidneys has been noticed two or three days after comminuted fractures. Fatty emboli may proceed from rupture of the liver, and from fatty metamorphosis of abscesses and of thrombi.

"The sudden entrance of a large quantity of air into the blood-current, such as may occur by incision of the large veins in the neck or near the heart, has been long known as the cause of rapid death. The fatal termination is due to the accumulation of the air in the right cavities of the heart, the contraction of which is unable to force the elastic air forward. The air remaining in the right auricle and ventricle forms an obstacle to the entrance of blood from the venæ cavæ, and arrests the pulmonary and systemic circulation. . . .

"While all arteries of the body are open for the reception of emboli, it is noteworthy that in certain situations the obstruction

of an artery by an embolus is absolutely harmless, whereas in other parts it is followed by characteristic structural and functional alterations. The main condition upon which this difference depends is the character of the arterial distribution in the various parts of the body. In certain organs and parts of the body the branches of the arteries do not anastomose with each other, communications existing only between the capillaries and between the veins. These arteries without anastomoses are called by Cohnheim *terminal* arteries (*Endarterien*). Such arteries are the renal, splenic, pulmonary, certain of the cerebral, and the central artery of the retina. The branches of the vena portæ also do not enter into anastomosis with each other. When an embolus lodges in an artery supplied with abundant anastomoses,—for instance, a muscular artery or one of the arteries of the extremities,—a collateral circulation is established, which prevents the part from suffering in its nutrition or function. The effect is widely different if no anastomoses exist between the peripheral part of the occluded artery and other vessels. In this case no arterial blood, or an insufficient amount, is sent to the capillaries of the parts supplied by the arteries. The part suffers in its nutrition, and may even undergo necrosis or death.

“An extravasation of blood sometimes, but not necessarily, supervenes in the district the arterial blood-supply of which has been cut off. These alterations constitute *embolic infarctions*, which are therefore of two kinds,—*white* or *anæmic infarctions* and *hemorrhagic infarctions*. An embolus produces a *white* infarction when its effect is anæmia and necrosis without hemorrhage. Anæmic infarctions are to be distinguished from decolorized hemorrhagic infarctions, with which they were formerly confounded. The primary and essential change in most white infarctions is *coagulation-necrosis*, the process by which the cells lose their nuclei and change their chemical composition. The details of this peculiar metamorphosis will be described under the passive alterations of the tissues.” (Flint.)

“Clots in peripheral veins, however small, are the sources of great danger. As a rule, they lead to secondary and multiple deposits and abscesses in the lungs; and it is chiefly differences in the size of the capillary vessels which determine their ultimate locality, where they act as any foreign body would. The *débris*

of clots, and large cell-elements from clots, in the mesenteric veins, and from ulcers of intestines, passing through the liver capillaries and proceeding to the lungs, where they are arrested, illustrate this. The lungs have the smallest capillaries of all. They average from $\frac{3}{10000}$ to $\frac{5.0}{10000}$ of a line (scarcely sufficient to let pass a white cell of blood or of pus, which on an average measures $\frac{4.4}{10000}$ of a line), whereas the liver capillaries have a much larger range,—namely, from $\frac{2.0}{10000}$ to $\frac{11.0}{10000}$ of a line.” (Aitken.)

“Specific infectious emboli induce pulmonary (so-called pyæmic) *abscesses*; non-specific emboli produce *hemorrhagic infarction*. Cohnheim states that a specific plug can never produce *both* infarcts and abscesses. This view is not taken by other observers.” (Loomis.)

“It is admitted as possible that thrombi formed in the left heart may break off, go the round of the circulation, and finally lodge in some branch of the pulmonary artery.” “Bed-sores, ulcerations, thrombosis of the femoral vein, phlegmasia alba dolens, wounds, and marasmic thrombosis are common peripheral sources of emboli.” (Loomis.)

HEMORRHAGIC INFARCTION.

“There have been various theories as to the source of the extravasated blood in hemorrhagic infarctions. The explanation advanced by Cohnheim, and derived from microscopical observation of the process in the tongue of the living frog, has been generally received. Occlusion by an embolus or by ligature of terminal arteries in the frog’s tongue is followed by cessation of the circulation on the peripheral side of the obstruction. Stasis occurs in the vessels in the district the artery of which is closed. The circulation goes on even with increased vigor in the surrounding vessels. The blood-pressure on the peripheral side of the obstruction being reduced to nothing, the blood flows back from the veins which connect with the capillaries the arterial supply of which is cut off. These veins receive their blood from surrounding veins from which the current has not been cut off. The capillaries of the obstructed district become filled with blood which has *regurgitated* from the veins. The walls of these capillaries and small veins, not receiving a fresh supply of arterial blood, finally become

weakened and allow a diapedesis of the red corpuscles. In this way Cohnheim explains the formation of embolic hemorrhagic infarctions. Recent experiments of Litten, however, seem to show that the extravasated blood, at least in some cases, is derived, not by return flow from the veins, but from the *adjoining* capillaries and small arterial anastomoses in which the pressure suffices to send the blood into the capillaries of the part whose artery is occluded, but not onward into the veins. Litten also maintains that many of Cohnheim's terminal arteries are such only in an anatomical, not in a functional, sense; that is, when they are occluded, blood may still reach their capillaries from surrounding capillaries and small arterial twigs, although the amount of blood may not suffice for the nutrition of the part." (Flint.)

We must again direct attention to the influence that terminal arteries exercise upon the pathology of hemorrhagic infarction and cerebral embolism. "None of the viscera,—and this observation belongs to Cohnheim,—where infarctus is not the rule, have the *terminal* mode of arterial distribution."*

In this connection I must remark that we cannot always absolutely affirm that anastomosis of the capillaries of the obliterated zones supplied by terminal arteries will not occur.

Duret, of France, and Heubner, of Leipsic, have contributed greatly to our knowledge of the minute anatomy of the cerebral circulation, but, unfortunately, they have not arrived at the same conclusions regarding the re-establishment of the collateral circulation in the so-called terminal arteries of Cohnheim.

I agree with Charcot that Heubner's facts are "*real*," but they are certainly "*rare*;" and Duret I believe is correct in stating that, "if the arteries of the encephalon are not *final* or *terminal* arteries, they very nearly approach that type.

"Heubner holds that the communications in question are very easy, that they are made by the mediation of vessels not less than a millimetre in diameter. He rests that assertion upon the results of injections, where he has invariably observed that the material injected into any one of the departments by a principal trunk, or by its branches, always rapidly penetrates the other territories. He also cites pathological cases which indicate that obliteration

* Charcot, Localization in Diseases of the Brain.

of one of the vessels of the cortical system or of its branches has during life given no evident symptom,—cases in which, death having followed, the cerebral pulp in the parts corresponding to the obliteration has at autopsy presented no trace of softening.”

Duret, on the other hand, says, “Let ligatures be placed upon each of the three principal arteries at the base of the encephalon on both sides, immediately above their origin in the circle of Willis. Then inject the Sylvian artery. This will first fill the Sylvian territory, and in the majority of cases it will pass beyond its limits. The injected material invades the neighboring parts slowly, little by little. This invasion is made from the periphery inwards towards the centre of the invaded territory. It is effected through the mediation of vessels of small calibre belonging to the system of *ramifications* having diameters of a quarter or a fifth of a millimetre, contrary to the opinion of Heubner, who holds that these arterial vessels have a diameter of one millimetre.

“The number of anastomoses from territory to territory are also quite variable. There are cases where one of the three grand territories can be injected isolatedly, the anastomoses not being sufficient to permit the injection to enter the adjacent territories. The communication which may occur at the periphery of a vascular territory explains why the obliteration of a main trunk often results in the softening of only the central parts of the territory, the peripheral portion remaining untouched.”

Charcot adds, “It need not be supposed that all obliterations of this kind would necessarily and surely produce such disastrous effects. There are *rare** cases where, in fact, such obliteration of a branch of the Sylvian artery, or even the artery itself (I here take the Sylvian artery as example; it would be the same for the *anterior* or *posterior cerebral* arteries)—there are cases, I say, in which the obliteration in question has no appreciable, or, at least, but passing, results.

“If this be so, it follows that the three main vascular territories into which the brain is divided, and the departments into which they in turn are separated, are not strictly isolated, individual territories. They may communicate, and indeed do communicate in the ordinary manner. But are these communications

* Italics my own.

easy and constant, or, on the contrary, are they accidental, indirect, and often impracticable? *In the solution of this question authors are at variance.*"*

"Infectious emboli, such as come from the cardiac valves in acute *ulcerative* endocarditis and from infected thrombi, produce effects entirely distinct from the mechanical obstruction to the circulation. They incite *suppurative* inflammation, and perhaps necrosis, wherever they lodge. Thus, even capillary emboli, when infectious, produce abscesses. The multiple abscesses in pyæmia are, for the most part, of embolic origin. Bacteria are generally to be found in infectious emboli, and, in some cases at least, constitute the poisonous principle." (Flint.)

Embolism in the brain is of frequent occurrence, and here we have to deal principally with its results. I have also told you that the embolus is generally lodged, for obvious anatomical reasons, in the *left* middle cerebral artery, in the fissure of Sylvius, resulting almost inevitably in softening, owing to the non-establishment of the collateral circulation, as the point of occlusion is situated above the circle of Willis. We have, therefore, no hope of recovery; for, should the patient not immediately perish, a hopeless hemiplegia will be the consequence.

There is a very important matter to which I wish once more to refer: it is the unfortunate use of the term apoplexy in connection with certain symptoms constituting the apoplectic state. Apoplexy, as commonly accepted, designates a particular condition (cerebral hemorrhage), and the synonymous use of the word is much to be regretted.

In a former lecture, when speaking to you of the apoplectic form of hyperæmia of the brain, I told you that the apoplectic condition is common to many different diseases. It consists in sudden abolition of consciousness, sensation, and voluntary motion.

This condition is present in cerebral hemorrhage; but it also exists in cases of epileptic coma, anæmic coma, uræmic coma, etc., and in fact in many diseases which possess very different pathological starting-points. Therefore, when we say that a man is in an apoplectic condition, we do not necessarily imply that he has cerebral hemorrhage.

* Chareot, *Localization in Diseases of the Brain*. Italics my own.

I will add that this apoplectic state not only *always exists* during the initiatory phenomena of cerebral embolism, but also constitutes a marked feature of the disorder. A decided anæmia will occur in the part which, by the obstruction of an important artery, is suddenly deprived of a large amount of blood. The consequence of this shutting-off of the vascular supply has already been considered,—the law being that the intensity of the disturbances will depend upon the more or less complete occlusion of the artery.

Hence, should the anæmia be complete, symptoms of depression alone will occur; if incomplete, we will observe an alternation of the symptoms of excitation with those of depression.

It is invariably of great importance to ascertain what disease has occasioned a *suddenly-developed* coma. Were the physician to confine his diagnosis to ascertaining the presence of *coma*, he would only expose his ignorance of the fact that this condition is but a *symptom*, and not a disease, and no more to be treated regardless of its pathological causation than would be a cough, a fever, or a dyspnoea.

The apoplectic condition does not occur in thrombosis unless the latter be very extensive; and in the majority of instances thrombosis is so gradual that the apoplectic phenomena set in only towards the *termination* of life, closing the scene. The reverse will be observed in embolism: the same symptoms manifesting themselves *ab initio*, being, in truth, the first signs which attract attention and cause the physician to be summoned.

The first knowledge, then, of the presence of embolism proceeds from the apoplectic state which overpowers the patient. When these apoplectic phenomena cease (which, however, does not always happen), what will be the condition of the patient? You will find a well-marked hemiplegia of the right side of the body (the embolism being generally in the left middle cerebral artery); and the reason that the paralysis is situated on the side opposite the one affected, is that the lesion is located above the point of decussation of the anterior pyramids of the medulla oblongata.

This hemiplegia is almost always permanent. Indeed, I have rarely seen or read of an instance where the paralysis completely disappeared after an embolism; the reason of this being that, when once plugged, the artery is closed forever.

A cork driven into the neck of a bottle will form an illustration, with this difference in the conditions: that in the case of the bottle the cork may be removed, but the embolus cannot be extracted from the artery.

It may be that, many years hence, at a very advanced stage of science, there will be discovered certain means by which to dislodge or dissolve the pernicious obstruction: this, though most improbable, we will not deem impossible,—agreeing rather with the late Dr. Dunglison, who, after a long and rich accumulation of scientific experience, “had learned never to use the word impossible.” But, to return to the subject, the patient, should he live at all, will remain hemiplegic, and the apoplectic phenomena will pass off. You ask me why the consciousness returns whilst the hemiplegia persists. Although I can give you no positive answer to this question, I believe, with Niemeyer, that the following explanation may be offered. The abolition of consciousness being due to the profound anæmia of the brain, collateral hyperæmia and resulting œdema follow, intensifying the apoplectic phenomena. After the lapse of a certain period of time, say a few days, this collateral œdema is re-absorbed, and, consequently, the apoplectic state, which was increased by the œdema, rapidly disappears; after which re-absorption, the pressure which was exerted upon the cortical cells of the convolutions of the brain is also relieved, these cells once more developing functions of ideation and of memory, and exhibiting intellectual activity and consciousness.

Another important fact to be mentioned in this connection is that collateral œdema following embolism is a condition not necessarily confined to the brain. Hence, if you know how to apply its phenomena to this organ, you will not be at a loss to apply them in any other situation. In the spleen, or the liver, or other organs, embolism is followed by collateral œdema. Why this collateral œdema follows embolism we do not positively know.

You will now ask, how is it that, the brain being a dual organ, one part should be rendered inactive, or even destroyed, and yet the other part remain without function? Why might not the right side compensate for the mischief created in the left? The answer is, that the collateral œdema is often extensive, and its

presence excessively deleterious, exerting more or less pressure upon the other hemisphere; and although, as I have told you, one of the functions of the falx cerebri is to prevent this encroachment, which it sometimes does successfully, in some instances it will be powerless in this respect, the falx then dipping over to the healthy side. The pressure, therefore, being transmitted, the dual action will be interfered with, and even temporarily abolished. It has been observed by Niemeyer that the posterior lobes are afforded better protection "against a pressure acting on the opposite hemisphere than the frontal lobes are, because the falx is much broader posteriorly and hangs much farther down than it does anteriorly."

We have still another important fact to consider: it is that we often have *coincident* embolisms. Of course it must be only after strong presumptive proof that you diagnose embolism; but if you want confirmatory evidence, examine the peripheral arteries that are easy of access, and you may sometimes find one or several of them obstructed. This fact will sometimes prevent you from being led into error. Remember, therefore, that coincident embolism may or may not be present. Coincident embolism is caused when the clot of fibrin constituting the embolus, after becoming detached, separates into several emboli, which are carried by the circulation into different arteries.

After having attentively listened to this lecture, you may still find it a very difficult matter to distinguish cerebral hemorrhage from cerebral embolism: only by thoracic exploration can you arrive at a safe conclusion. Let us imagine two cases both at the same time falling into the apoplectic state. The diagnosis will be based upon: 1st, a *history of the cases*; 2d, the examination of the chest; 3d, the sequence of events; 4th, the age of each individual. The age is very important, for we know that cerebral hemorrhage sometimes results from atheromatous arterial changes, frequently themselves an accompaniment or a cause of thrombosis, which is a disease peculiar to old persons. Embolism, on the contrary, occurs at all ages, irrespective of sex or of other conditions of life. Yet in some instances obscurity exists, age not settling the question. In this disease, and in others, typical cases do not always solve the problem. It is known that even children are occasionally victims of cerebral hemor-

rhage. The rule, however, is that in cerebral hemorrhage age has great significance,—the disease usually affecting persons past the meridian of life, being frequently the result of *endo-arteritis deformans*.

We may now sum up the points of diagnostic difference between thrombosis and embolism :

| THROMBOSIS | EMBOLISM |
|--|---|
| Is generally preceded by brain-symptoms. | Is rarely preceded by brain-symptoms. |
| Symptoms gradual. | Symptoms sudden. |
| No rheumatic history. | Oftentimes rheumatic history. |
| Occurs in advanced age. | Occurs at all ages. |
| <i>Endo-arteritis deformans</i> of peripheral arteries sometimes occurs. | Coincident embolisms are sometimes present. |
| Apoplectic phenomena during the last stages. | Apoplectic phenomena <i>ab initio</i> . |
| Is always formed <i>in loco</i> . | Proceeds from some remote point of the circulation. |

Never take any one symptom as a positive evidence of a disease. Do not confide too strongly in typical descriptions. I have never seen a case of essential fever, or any other malady, which strictly corresponded in every particular with the classical delineations of authorities. You must rely greatly upon the care you exercise in studying disease, and upon your earnest efforts to diagnose properly. Do not essay more than you can accomplish, thereby becoming careless, and prescribing for various symptoms without arriving at their proper interpretation. Exact diagnosis underlies all rational therapeutics.

You cannot always leave the bedside perfectly satisfied that you have mastered the problem of your patient's ailment; the most accomplished practitioner sometimes fails to ascertain in one visit the true cause of a malady, and, indeed, several visits may be made before certain diseases can be recognized, if recognized at all. Again, I am satisfied there are some affections which cannot be brought under any nosological arrangement. Hence you should endeavor to become good diagnosticians, and, when you are unable to classify a disease, should apply to its treatment the principles derived from your general pathological and therapeutical resources.

Should you visit a patient for the first time in consultation

with another physician, do not let him imbue you with his own ideas before making an examination for yourself, or you will almost certainly be biassed.

I feel happy to state, gentlemen, that these views of Niemeyer as to the pathological anatomy of partial cerebral anæmia, which I have taught to successive classes for over twenty years, have recently been confirmed by Charcot.

CHARCOT'S MOST RECENT "CLINICAL AND ANATOMICO-PATHOLOGICAL RESEARCHES UPON CEREBRAL SOFTENING AND ENCEPHALITIS." *

"Anatomical study of *cerebral softening* in old men has led Charcot to affirm the correctness of the doctrine which has taught the dependence of this affection upon alterations of nutrition due to circulatory disturbances, and this equally in all regions of the brain. Modifications of the cerebral circulation are produced by arterial embolism, atheroma, arterial thrombosis, or thrombosis of the cerebral sinuses; and in all cases the softening is the result of a retrogressive transformation of the nervous tissue; it is emphatically not an inflammatory process."

Although the consideration of *encephalitis* must be postponed to the second volume of these Lectures, I desire in this connection to refer to certain observations of Charcot, which are intimately related to the subject of cerebral softening and encephalitis.

Charcot claims that "in encephalitis the anatomical alterations are primarily due to a proliferation of the cellular elements. In softening, the granular fatty infiltration of the cerebral substance is the first appreciable lesion, which is recognizable twenty-four hours after the vascular obstruction. Sometimes the elementary granules are found isolated, sometimes in round masses, constituting a variety of granular corpuscles. Hence there exists, from an anatomical point of view, a fundamental distinction between cerebral softening and encephalitis." This distinction is not unimportant from a clinical stand-point. It is corroborated by a great number of observations made at the same time by Vulpian and Charcot,—namely, that certain precursory phenomena of softening, such as mental confusion, are more related to cerebral

* Œuvres Complètes, 1890.

ischæmia than to cerebral congestion, and that certain symptoms which are generally ascribed to encephalitis, such as delirium, convulsions, contractures, are very rare as initiatory phenomena of softening; when observed in the latter, they are usually to be referred to some complication. Late contractures, so frequently encountered in the paralyzed limbs of individuals affected with old softenings, are the result of a consecutive lesion of the spinal cord (descending sclerosis). Charcot concludes his remarks on this subject by citing the confirmatory opinions of Messrs. Prevost and Cotard, also referring to an inaugural thesis of M. Poumeau, and stating that M. Proust in a thesis cites some facts relating to this subject communicated by M. Charcot to him.

Charcot has also directed attention to a "kind of softening, not unusual, which occurs in the *cachexia* of cancer as a result of arterial thrombosis from inopexia."

SYMPTOMATOLOGY OF ANÆMIA FROM COLLATERAL ŒDEMA.

How may we recognize anæmia as the result of a *collateral* œdema in the brain? In order to do this, we must be guided by the following facts. Whenever we have a partial anæmia, the result of a collateral œdema, the symptoms of one will be complicated by those of the other. We have seen that collateral œdema is sometimes due to embolism; yet, again, we know that hyperæmia of the brain will sometimes have the same effect. In collateral œdema following embolism, a capillary anæmia will be produced, which, if not relieved, may terminate fatally. Furthermore, when a collateral œdema, the result of hyperæmia (which has not yet resulted in permanent partial anæmia), occurs, we will find the symptoms of collateral œdema accompanied by those of the *primary* disease.

All the pathological variations already referred to—partial hyperæmia, collateral œdema, partial anæmia, softening, etc.—are produced by tumors, clots, abscesses, or anything that will persistently encroach upon the substance of the brain or disturb its circulation. When the symptoms of intense hyperæmia present themselves, no matter by what cause produced, the secondary phenomena will be readily understood, and the ultimate result may be softening of the brain. Now, when you have a patient presenting these symptoms, with a violent collateral œdema of the

brain, will you attack that œdema with diuretics, or absorbents, or derivatives? Most assuredly not; but you will endeavor, by directing your attention to the *primary* affection, to relieve the hyperæmia, by which you may prevent softening. In other words, treat your patient anti-congestively, so to speak, and you will at least palliate, should you not cure, the disease. But if, by an unpardonable oversight, you were to treat certain symptoms, perhaps those of the secondary anæmia, you would of necessity make matters worse; for you would thereby intensify the hyperæmia, and a hopeless softening would surely result. You therefore readily understand, and of course appreciate, the necessity of dwelling upon so important a point. Hence remember that, should you diagnosticate a secondary anæmia resulting from a previous brain-disease, or from anything else imaginable, the symptoms will be blended with those of the primary affection, whatever that may be, which it is your business to be able to recognize and to treat accordingly.

This collateral œdema enables us to explain several obscure forms of morbid manifestations; for instance, it must have occurred to those of you who have witnessed post-mortem examinations that the appearance of the brain is sometimes quite different from what we expected to find, judging from the symptoms present during life.

Let us suppose, for example, that you have a patient suffering from hemiplegia, a paralysis either of motion or of sensation, or of both, with or without spasms, etc. After death you examine the encephalon, and all you find may be a small clot lying upon the superior part of the periphery of the brain and not necessarily in the motor tract, or a disease which does not encroach on the cranial cavity. Now, how will you proceed to explain that a very small clot, situated upon the surface of one of the convolutions of the brain, may under these circumstances produce paralysis? You will answer, by the pressure the clot exercised upon the convolutions, which pressure is transmitted to the medullary fibres, and finally to the corpus striatum, or even to more distant parts at the base of the brain. Such, however, would be an incorrect conclusion; for, if you recall what I have already stated, you will remember that the brain is incompressible.

You have not, I am sure, forgotten the explanation of Niemeyer,

that a disturbance so situated may produce an irritation, in the neighborhood of which you may have a hyperæmia, and, as a result of this, a collateral œdema, and finally anæmia; and if this collateral œdema dip deeply into the substance of the brain, and the *transuded serum* press upon the corpus striatum or internal capsule, a paralysis may be the result.

In this manner, then, are we enabled to explain how a minute clot upon the periphery of the brain may produce paralysis; and in proportion as the peripheral clot becomes absorbed, the resulting hyperæmia will be removed, the collateral œdema disappear, and the symptoms of paralysis slowly and gradually pass away.

In addition to the foregoing, we have symptoms of other diseases, which can be explained only by the pressure of collateral œdema. For instance, the cerebellum may be partly destroyed without any peculiar symptoms necessarily revealing the fact during the life of the patient. (It has been contended by eminent physiologists that the cerebellum is the seat of the power of co-ordination; but this I am by no means disposed to admit.) On the other hand, it happens that some of the most serious symptoms, such as hemiplegia, sometimes coexist with the above lesion.

How are we to understand such paradoxical conditions? How shall we explain these seeming contradictions? The enigma is solved when we recollect that we may have a collateral hyperæmia extending upward towards the brain, and followed by a collateral œdema. In the former case, a hemiplegia would be explained. The hyperæmia may extend towards the corpora quadrigemina by the superior peduncles of the cerebellum, the *processus e cerebello ad testes*; or it may be conducted along the middle peduncles, reaching the pons Varolii or the tuber annulare; or, again, it may travel along the inferior peduncles to the restiform bodies, producing, in each case, corresponding phenomena, which may exist separately or conjointly, causing the most serious and complicated results.

We have but one more form of symptoms to consider in connection with anæmia of the brain: any force or foreign body or any imaginable pathological condition that will in any way encroach upon either hemisphere or the cerebellum may produce disturbances of the circulation, and result in paralysis, hemiplegia,

softening, etc. Should the pressure be on one side, we will usually have a hemiplegia of the opposite side of the body. Should a tumor or an abscess be present, the paralytic phenomena will probably be slowly produced, because there will be sufficient time for a gradual atrophy of the brain to prevent great encroachment on the surrounding mass on the part of these adventitious products. But the medullary masses of one hemisphere may be diseased and softened by tumors, abscesses, etc., and still no marked symptoms necessarily result. This is due to the *comparative* non-importance of the former and to the compensatory action of the opposite hemisphere, and to the growth of the latter having been very slow, with a corresponding absorption of brain-substance as it became replaced by the intruding body. But in anæmia, which occurs when the tumor, etc., develops rapidly, in proportion to the amount of destruction of the nerve-cells, you will find symptoms of depression speedily developed and brain-disturbances of a paralytic character. It is important to remember that these phenomena will depend greatly upon the situation of the parts involved. Were there no perplexing difficulties in the recognition of symptoms peculiar to diseases of the brain, their study might become more satisfactory. Experience teaches us that the diagnosis of all brain-maladies is quite obscure. There is one condition, however, the symptoms of which are, according to Niemeyer, unmistakable, almost pathognomonic, so that we are enabled to diagnosticate it quite accurately. I allude to an *encroachment* of some kind upon the *posterior cranial fossa*, that part which lies beneath the tentorium cerebelli. The symptoms attending diseases of the posterior cranial fossa are: 1st, pains in the occipital region; 2d, vomiting; 3d, a morbid impairment of sensibility (incomplete anæsthesia); 4th, a peculiar development of partial paralyses; 5th, dysphagia and impaired articulation; and 6th, *dizziness possessing certain distinctive peculiarities*. We all know that dizziness may be the result of an indigestion, or of an over-indulgence in fermented or distilled liquors, or may proceed from various causes existing wherever vertigo is present, whether originating in the stomach, brain, or other part. But in these last-mentioned cases the dizziness is always subjective, being a hallucination,—patients imagining that surrounding objects are continually whirling around them. Whether the

patient is lying down, standing erect, or walking, the dizziness will continue without interruption ; while, on the other hand, *in diseases contracting the space in the posterior cranial fossa this dizziness exists only when the patient is walking, moving, or in the erect posture, and immediately disappears on his lying down.*

After this dissertation upon partial anæmia, you would undoubtedly like to be compensated with practical observations regarding the best modes of its treatment, and a careful comparison of their relative values. In this respect I must disappoint you, for the most I can say is, that *the less you actively interfere, the better!* Thrombi and emboli, you are aware, cannot be mechanically removed ; nor have we any therapeutical resources at our command with which to attain such an end. Several things—stimulants, for instance—have been recommended, but they are all useless ; for they can never unplug an artery. Nevertheless, you must not fail to resort to the best hygienic treatment possible, using tonics to counteract loss of vigor. You may also treat symptoms as they arise, and, above all, must endeavor to obviate collateral hyperæmia.

Although a part of your patient's brain may be anæmic, death from a collateral hyperæmia may occur. Should the symptoms of depression, which always exist in profound anæmia, be complicated by alarming symptoms of irritation, beware of collateral hyperæmia, and treat your patient accordingly, blistering, leeching, purging, etc.,—with due caution, however,—and you will at least succeed in averting an immediate and perhaps transitory danger.

LECTURE IV.

GENERAL CEREBRAL ANÆMIA.

Symptoms—Diagnosis—Treatment.

GENTLEMEN,—In order more fully to understand the physiology of cerebral anæmia, let us for a moment consider the requirements of the nervous system and the conditions of its normal excitability. They are :

1. Constitutional integrity of nerve-substance.
2. Normal circulation of the blood.
3. Normal composition of the blood.
4. Alternations of rest and activity. (Jaccoud.)

To these we may add *a normal temperature of the blood and perfect vascular integrity.*

At this juncture it is well to recall what has been said in a preceding lecture, that the symptomatic phenomena of cerebral hyperæmia and anæmia do not in any respect differ from one another, if the symptoms alone are considered.

It is well to bear in mind that cerebral anæmia, when the *quality* of the blood is impaired, is always of *gradual* occurrence, and affects the entire brain, but when the *quantity* of blood of the whole brain is deficient its abstraction may be either *sudden* or *gradual*.

Pressure on the blood-vessels going to the head, and aortic aneurisms, are sometimes the causes of diminished blood-supply to the brain.

The operation of paracentesis abdominis may sometimes produce a comparatively bloodless condition of the brain, which may terminate fatally. Associated with this there is often an unequal distribution of blood, which peculiarly leads to a dilatation of the intestinal vessels when dropsical accumulations are suddenly removed.

Spasm of the blood-vessels of the brain with accompanying

narrowing of their calibre, produced by various causes, is a well-known factor in the production of sudden and transitory anæmia, exemplified in the intense cerebral anæmia superinduced by capillary contraction during the initiatory phenomena of epilepsy.

Pathological conditions of imperfect blood-renewal play a prominent rôle in the production of cerebral anæmia.

Fevers have most pernicious effects on the constitution of the blood, and develop and intensify anæmia by tissue-oxidation and corresponding retrograde metamorphosis, to say nothing of the retention of effete or excrementitious material in the circulation.

These pernicious results and qualitative perversions of fevers, inflammations, and other acute maladies upon the blood are familiarly evinced during convalescence from protracted disease, and by the "delirium of inanition," described by Dr. Clymer so graphically in Aitken's Practice.

General cerebral anæmia most frequently originates in causes not restricted in character, but which are expressive of particular states of the system at large. All *hemorrhages*, excessive or colliquative discharges, whether *menstrual*, intestinal, hemorrhoidal, uterine, suppurative, or exudative, but especially the first mentioned, which are by far the most common, must sooner or later induce an anæmia, more or less profound, from which the brain will suffer, entailing thereby a disordered and imperfect performance of its functions. The physiological law already referred to must not, in this connection, be lost sight of. If the cause capable of resulting in abolition of the nervous excitability be not sufficient to do so at the commencement, its partial effect will invariably be a precedent condition of *exaggerated functional activity*. This furnishes an explanation of many of the phenomena of cerebral anæmia, and offers the only rational interpretation of the commingling of the symptoms of irritation with those of depression which is common to this disease, and which leads inexperienced physicians to suppose that they are dealing with congestion, which they treat actively and with disastrous results, while, in reality, anæmia, more or less profound, lies at the bottom of the difficulty. *The hydrocephaloid of Marshall Hall* exemplifies what we desire to designate as a common source of error in this respect, its etiology being frequently misunderstood, while therapeutic re-

sources directed to its relief, from their inappropriateness, often increase the evil they are intended to avert.

In cases where cerebral anæmia is very gradually produced, an exaggeration of the excitability of the brain will result in what Gowers calls "*irritable weakness*." The cerebral activities will then be excited with abnormal facility. Convulsions themselves are no indication of a different pathological condition: indeed, as Gowers adds, "that the nerve-cells should 'discharge' when the blood-supply is arrested is a fact of very great physiological interest as an indication of the *reserve* of force that must be stored up in the nerve-cells, and of the probability that sudden overaction is due to *diminution of resistance to action*, and not to an increase in the force-generating function of the cell.* Latent energy may be liberated, but new force can scarcely be produced under the influence of sudden anæmia."

It must be remembered that anæmia during life is not proved by the mere pallor of the brain as observed on autopsy, as the modes of death greatly influence the amount of blood found in the brain in post-mortem examinations.

In diffused cerebral anæmia much cerebro-spinal fluid is often present, and alters the appearance of the nerve-cells, and not infrequently develops many of the conditions which we found existing when describing the anatomical appearances of collateral œdema. If these conditions persist, irreparable damage may be done to the nutrition of the brain.

Quantitative and qualitative blood-changes will more or less materially influence the production of cerebral anæmia, especially when, from chylipoietic disease, systemic conditions, or the different dyscrasias, improper or deficient hæmatosis results. The absence of properly arterialized blood—its presence being a *sine qua non* for the due performance of the cerebral functions—will be readily appreciated.

It follows, from what we have said, that anæmia is produced whenever the brain receives an insufficient quantity of blood, whether this be due to arterial obstruction or to reduction of the volume of this fluid. Moreover, the oxygen of the vital current, being an essential agent, must not be unduly diminished,

* Italics in this quotation are my own.

for the proper nutritive processes would not be accomplished, and the normal performance of their functional activities would be prevented. The red corpuscles being "carriers of oxygen," a deficiency on their part, so far as the brain is concerned, would be equivalent to inadequacy of the blood itself. Jaccoud and Niemeyer mention, among the causes of general cerebral anæmia, those of vascular origin, often reflex in character, produced by unusual mental emotions, eventuating in a contraction of the cerebral vessels. In these cases, loss of consciousness and sudden pallor of the countenance constitute the prominent symptoms.

Mechanical causes are also cited, as in instances where an undue amount of blood is retained in other organs at the expense of the general circulation. It is a well-known fact that syncope is frequently caused by *moving patients ill with fever*, or by patients assuming too soon the erect posture while convalescing from protracted and exhausting affections. Dr. Todd, in his work upon Acute Diseases, particularly insists upon the serious dangers attendant upon exertion in both instances. The hazard is enhanced by a combination of such causes as an enfeebled cardiac impulse and the sudden accumulation of blood in the lower extremities.

The symptoms will vary, according to the slow or rapid development of the anæmia. In the latter instance, syncope—perhaps accompanied by convulsions, if the anæmia be intense—may be anticipated. In cases of slowly-produced and more permanent cerebral anæmia, the symptoms of irritation will be more or less prominent. In graver conditions of this pathological state, marked symptoms of depression will succeed those of excitation, which are merely temporary and initiatory. These latter will so closely resemble the phenomena presented by cerebral hyperæmia as to be inseparable in description; while the former will be more particularly distinguished by such expressions as "syncope" and "physical and intellectual apathy."

DIAGNOSIS.

The symptomatic manifestations of hyperæmia and anæmia are identical, and furnish no clue by which we can recognize and differentiate these two conditions of diametrically opposite pathological character. Maudsley says that "since the time of Hippocrates it has been known that when there is too little blood in the brain,

symptoms are exhibited similar to those which are produced by a *congestion* of blood: pains and swimming in the head, confusion and incapacity of thought, affections of the senses and of movement, occur in consequence of anæmia of the brain as certainly as they do in consequence of congestion. In both cases the due nutrition of the nerve-cell, which is the agent of cerebral function, is greatly hindered; and much of the ill effect is similar, though the causes appear to be so different. In reality, however, the causes are not so different when we proceed to analyze the conditions comprised under the terms anæmia and congestion. In that continued relation between the organic element and the blood by which the due reparative material is brought and waste matter carried away, it amounts to much the same thing whether through stasis of the blood the refuse is not carried off and reparative material brought to the spot where it is wanted, or whether the like result ensues by reason of a defective blood and deficient circulation; it is little matter to the inhabitants whether the street is almost blocked, or whether its entrance is almost closed, so long as free circulation is prevented." The history of the case, concomitant symptoms, and general condition of the patient's system will therefore furnish the only reliable data upon which to base our conclusions, which would necessarily be erroneous were we to rely *exclusively upon the cerebral symptoms*, or even attach undue importance to the appearance of the patient; having before stated that pallor may coexist with profound and dangerous hyperæmia, thus constituting a source of fallacy not to be forgotten. The clinical, pathological, and therapeutical antecedents must, therefore, be carefully studied. I can recall a case in reference to which I was for a few days in doubt, but finally diagnosticated cerebral anæmia, because the lady was greatly relieved when, in lowering her head, she favored the gravitation of the blood to her brain, and all her symptoms became remarkably exaggerated when in the erect posture. You who were with us last winter will recollect the successful issue of a case which I diagnosticated to be general cerebral anæmia, at the hospital, the result of a profuse hemorrhoidal flow, accompanied by epistaxis, which rapidly yielded to iron, digitalis, and a liberal administration of nutrients. Yet, owing to some striking symptoms of excitement, the case had previously been treated by several experienced physicians by spo-

liative measures, on the supposition of intense cerebral hyperæmia existing, with the result, I need hardly add, of greatly aggravating all the symptoms, and endangering the patient's life. Cardiac examination must never be neglected in doubtful cases, as an enfeeblement of the first sound or a diminished impulse, especially if accompanied by blood-murmurs, would have the utmost significance.

All conditions of anæmia are exaggerated in the erect posture. "It has been remarked that some anæmic persons can think well only when lying down." (Gowers.)

The mode of meeting the indications of a cerebral anæmia resulting from a sudden and profuse loss of blood resolves itself into the usual modes of relieving syncope. Position, arterial compression, even the temporary application of the tourniquet upon the principal superficial arteries, Nélaton's method used in chloroform narcosis, consisting in holding for a long period the lower extremities high above the patient's head, the preparations of ammonia, ether, and brandy, internally administered (or the two latter hypodermically given), and, in very critical cases, transfusion, should all be judiciously essayed. In cases of habitual anæmia, arsenic, chalybeate preparations,* the improvement of the nutritive functions, an easily-digested and liberal dietary scale, especially of milk and highly-nitrogenized substances, and the removal of the cause of the pathological condition, where possible, are resources of the greatest value, and will often be rewarded with success.

In conclusion, I would particularly admonish you, gentlemen, to bear in mind the dangers of *hydrocephaloid* disease in young children. Treat their diarrhoeas—a fruitful source of this affection—early and earnestly. Do not refrain from arresting the intestinal discharge for fear of producing brain-symptoms, as is too often done, in compliance with a maternal prejudice, fraught with danger to the little ones. Recollect, as Trousseau says, "that the continuance of diarrhoea in teething children predisposes to convulsions." Do not be deluded by the supervention of the symptoms of irritation in children who have experienced colliquative

* My experience has conclusively proved that the salts of manganese are often advantageous when combined with, or even substituted for, those of iron.

discharges, notwithstanding the flushed face, heated skin, general restlessness, twitching, insomnia, and even convulsions. In such subjects, resort to stimulants and milk, and, in older children, to the famous "raw meat diet." You will thereby prevent the subsequent stage of collapse, when the vital powers will be too prostrated to admit of recuperation.

Forewarned, you should be forearmed; and it will henceforth be inexcusable in you to commit such a blunder as to treat similar cases by spoliative measures. Resist all temptations to be misled by the threatening aspect of the initiatory symptoms, so deceptive as to compel you to select therapeutic means which would inevitably result fatally, consigning to a premature grave the little sufferer committed to your care; which catastrophe, instead of being averted, would be precipitated by an ignorance as unpardonable as unjustifiable.

An elegant formula for the administration of manganese is that of Wyeth's "compound syrup of the phosphate of manganese," which also contains phosphate of iron, the dose being a teaspoonful three times a day after meals, to which two or three minims of Fowler's solution of arsenic to each dose may be added in appropriate cases. When it is desirable to administer a preparation of iron which does not constipate, the following formula will be found excellent:

R Tinct. ferri chlor., fʒiii;
 Liq. ammon. acetatis, fʒi;
 Acidi acetici dil., fʒii;
 Syrupi zingiberis, ad fʒiv.—M.
 S.—Two teaspoonfuls ter die after meals, in water.

In cases where there is a malarial complication the following pill will be found valuable:

R Quin. bisulph., gr. c vel ʒi;
 Ext. nucis vom., gr. vii;
 Acidi arseniosi, gr. i;
 Pilulæ ferri carb., ʒi.
 M. et ft. pilulæ no. xx.
 S.—One ter die after meals.

The amount of quinine above recommended can be varied, so as to make each pill contain either three or five grains of quinine.

Another very powerful tonic pill which I use in many of these cases is composed as follows :

R Strychninæ sulph.,
Acidi arseniosi, aa gr. iv ;
Ferri sulph. exsicc., gr. c ;
Quininæ sulph., gr. c ;
Ext. digitalis, gr. xvi.
M. et div. in pilulas no. c.
S.—One pill three times daily after meals.

A pill which I originated many years ago, and which has been thoroughly tested, having indeed proved invaluable in many cases, especially in general anæmia, chlorosis, hysteria, melancholia, hypochondriasis, and neurasthenia, is composed as follows :

R Auri et sodii chlor., gr. v ;
Strychninæ sulph., gr. iiss ;
Ferri arsen., gr. xvi $\frac{1}{2}$;
Quininæ sulph., gr. c.
M. et div. in pilulas no. c.
S.—One ter die after meals.

Most of these pills are made for me by McKesson & Robbins, of New York City, and Sharpe & Doane, of Baltimore. I often add to this treatment the various preparations of maltine made by the Maltine Manufacturing Co., of New York. I have for years used the latter as a more easily assimilated nutritive agent than cod-liver oil ; the last-named article I prefer to give in emulsion :

R Ol. morrhue opt., f 3 vi ;
Pulv. acaciæ, 3 vi ;
Aq. destil., f 3 ii ;
Glycerol. hypophosph.,
Syrupi calcis lactophosph., aa f 3 iv ;
Ol. gaultheriæ, mxx.—M.
S.—One tablespoonful three times daily after meals.

I often use modifications of what I call "nerve-food" in cases of anæmia and neurasthenia. One of the combinations of this character that I most frequently use is composed of equal parts of glycerole of the hypophosphites and syrup of the lactophosphate of lime, the dose of which would be two teaspoonfuls before meals. I vary this sometimes as follows :

R Syrupi calcis lactophosph.,
 Glycerol. hypophosph.,
 (Fairchild's) Ess. pepsin. vel elix. peptonoids,
 Elix. calisayæ, aa fʒi.—M.
 S.—One tablespoonful ter die before meals.

I find the addition of pepsin to this combination valuable where gastric disturbances exist.

Phosphorus may also be administered, in pills of phosphide of zinc and extract of nux vomica, as recommended by Hammond,—viz. :

R Zinci phosphidi, gr. iii ;
 Ext. nucis vom., gr. x.
 M. et ft. in pil. no. xxx.
 S.—One ter die.

I sometimes use phosphorus in the form of Thompson's solution, as recommended by Seguin. However, this preparation is an exceedingly strong one, and is more applicable to intractable cases of neuralgia. I agree with Hammond that nitrite of amyl by inhalation is highly beneficial in cerebral anæmia, although I believe the most useful application of this remedy is to abort the aura epileptica. I will, however, refer more particularly to the uses of nitrite of amyl and Thompson's solution of phosphorus in my lectures on epilepsy and neuralgia.

Whilst alcohol and opium constitute powerful remedial agents in treating all cases of anæmia, I emphatically protest against their careless and indiscriminate administration. *I have for years taught my students that there is a great responsibility in resorting to them in all chronic cases, as their improper and habitual use but too frequently morally wrecks the patient, who thus becomes the innocent victim of professional carelessness.* In fact, a maxim with which I permit no compromise interdicts the use of opium and all its preparations, and of all vinous, malt, and alcoholic stimulants, in chronic diseases. Physicians cannot be too strenuously warned in this direction. I know but one exception to this rule, which is the classic use of opium in melancholia: under these circumstances patients should be kept ignorant of the nature of the drug they are taking, and the prescription should *not be renewable* without the express permission of the physician.

In all cases where diet is an important adjunct of treatment,

a distinct diet-list should be furnished to the patient, who should be allowed no discretion in this matter. Just as there exists a dosage in electricity, there should be correspondingly one of exercise, and a dosage, as well as scientific regulation, in matters of diet.

Where the patient is much prostrated, neurasthenic, and suffering from a want of *muscular tone*, I have found the following prescription of Prof. Frank Fry, of this city, invaluable :

R Ext. cocæ fl., fʒiii;
Tinct. nucis vom., fʒiii;
Elix. simplicis, q. s. ad fʒiv.—M.
S.—fʒii ter die before meals.

Inasmuch as a proper performance of the functions of the stomach, and the healthy assimilation of food dependent thereupon, are essential in maintaining a proper nerve-tone, it becomes a matter of primary importance, in the treatment of all cases of functional nervous disease, to obviate dyspeptic tendencies; otherwise a vicious circle of morbid factors will become established: the digestion being impaired will result in faulty nerve-nutrition, and as a direct result thereof defective innervation of the digestive organs will inevitably ensue. To prevent such evil consequences I have by a long experience found the following combination, coupled with a well-regulated diet, most effective. The charcoal may be added or omitted, in accordance with the presence or the absence of *flatulency*.

R Lactopeptine,
Boudault's pepsine,
Willow charcoal,
Bismuthi subnit. (French), aa ʒiiss;
Aque, fʒi;
Elix. calisayæ, fʒiiss;
Tinct. cardamom. comp., fʒiiss;
Jensen's pepsine, gr. xx.—M.
S.—One teaspoonful after meals.

The regulation of the bowels by laxatives is an indispensable feature of treatment. Thirty years ago, based upon Schroeder van der Kolk's long and successful experience with the drug, I introduced the use of buckthorn bark (*Rhamnus Frangula*) in the West; and I have been charmed with its effects, finding that

in every respect it answers the purposes for which it has been so highly extolled. Van der Kolk originally claimed for it the following qualities. In the first place, it is a tonic, and although it does not belong to the family of gentians it acts in a very similar manner. In the second place, it is also stomachic and carminative. In the third place, it is the only known laxative whose long-continued use permits of the reduction instead of the increase of the dose. In the fourth place, it simply increases the alvine actions, without any debilitating effect. To use his own words, "it has the peculiarity of securing a solid evacuation without inducing griping or pain, and at the same time it has no nauseous taste. It does not give rise to colic. The medicine is peculiarly suited for a long-continued employment."

Van der Kolk prescribed it in decoction. I have preferred the fluid extract, made as follows: bark of *Rhamnus Frangula*, two pounds; water, eight pints; boil until reduced to fifty-six fluid-ounces; express and strain; add alcohol eight fluidounces. Adult dose, two teaspoonfuls at bedtime, as a laxative.

In severe cases of constipation, when this fails, I use the following pill:

R Ext. aloes aquosi, gr. lx;
 Ext. colocynth. comp., gr. xx;
 Ext. hyoscyami,
 Fel. bovis, aa ℥i.
 M. et div. in pilulas no. xx.
 S.—One at bedtime when necessary.

I have very frequently prescribed with satisfactory effect in these cases, at bedtime, a teaspoonful of pulvis glycyrrhizæ comp. (Prussian Pharmacopœia). In cases where *headache* exists, whether of neuralgic, neurasthenic, or migrainous origin, I have rarely known the first dose of the following mixture to fail to produce immediate relief of the cephalalgia:

R Antipyrin., ℥iv;
 Caffein. citrat., gr. viii;
 Tinct. strophanthi, m. xv;
 Elix. potass. bromidi, f℥i;
 Ammon. muriat., ℥ii;
 Elix. guaranæ, q. s. ad f℥ii.—M.

S.—Two teaspoonfuls every three hours until relieved or until three doses have been taken.

The cold sponge-bath, massage, the continuous galvanic current, general faradization, and *central* galvanization are invaluable auxiliary measures of treatment when judiciously and scientifically administered.

Nitro-glycerin has been highly extolled in the treatment of anæmia. I fully agree with Bartholow when he remarks that "it should be understood, also, that the improvement of nutrition by increased alimentation is the more complete because, by the action of nitro-glycerin, a much larger quantity of blood is obtained by the tissues, and hence more of the nutritious matters, than would otherwise be available. The author has availed himself of these facts, and has utilized nitro-glycerin in the treatment of *anæmia* in its ordinary form, and in the *pernicious* variety. One of the most common of the therapeutical fallacies of the day is the giving of iron to cure anæmia, for in a large proportion of the cases the iron cannot be assimilated. The organs concerned in blood-making may be in a pathological condition or functionally torpid. Stomachal and intestinal digestion may be in such a state that the ordinary preparation of food-stuffs is too imperfectly performed for the materials to be utilized in blood-making. To cure anæmia something more is requisite than to give iron. The functional or pathological states that interfere must be removed. When all the digestive and assimilative functions are restored, failure is still encountered by imperfect distribution of blood. The heart may be feeble and act imperfectly, the peripheral arterioles may contract on their lumen, and thus hinder the passage of the blood. Such is the condition in a large proportion of the cases of anæmia. To bring about a proper activity of the nutrition, it is necessary to restore the organs of circulation, and admit the fullest nutrient supply to all the tissues. It is this function of trinitrin that places it in the front rank of remedies for anæmia."

LECTURE V.

MENINGITIS.

Acute Idiopathic Meningitis, or Leptomeningitis—Pachymeningitis—Tubercular Meningitis—Cerebro-Spinal Meningitis—Simple Idiopathic Meningitis—Chronic Meningitis—Characters—Symptoms: Chill, Fever, Headache, Delirium, Vomiting, Constipation—First Stage—Second Stage—Pericarditis—Pneumonia—Rheumatism—Typhus and Typhoid Fevers—Syphilis—Hydrocephaloid—Prognosis—Causes—Convulsions in Children—Treatment: Drastic Purgatives, Cold Applications, Ergot, Bromide and Iodide of Potassium, Vesicants, Venesection, Leeches, Cupping, Counter-Irritants.

GENTLEMEN,—We will now consider a class of affections of which delirium is always a prominent symptom, adopting Da Costa's method of grouping the nervous diseases according to some particular common symptom. Among the numerous acute affections of the brain which delirium always helps to characterize (being almost one of their pathognomonic symptoms), we have—

1st. Acute Idiopathic Meningitis, or Leptomeningitis. 2d. Tubercular Meningitis. 3. Cerebro-Spinal Meningitis. 4th. Chronic Meningitis.

In a consideration of these diseases it is not necessary to fully develop all their details, as we have reviewed many of them with due care when speaking to you of hyperæmia and anæmia.

We therefore have only to apply the laws established, making but slight reference to facts already fully explained.

The disease which I will place before you to-night is that form of meningitis called

LEPTOMENINGITIS.

The term leptomeningitis signifies an inflammation of the soft membranes enveloping the brain (the pia and the arachnoid). These membranes are called meninges; and by affixing *itis*, which denotes inflammation, the word meningitis is formed (as in bronchitis, pleuritis, etc.).

Meningitis presents a few peculiarities, as the following :

The inflammation may be limited to the convexity of the membranes, as in acute idiopathic meningitis; or it may attack the base, as in tubercular meningitis; or the dura may be the only membrane implicated, as in pachymeningitis, or the pia and the arachnoid may be the involved membranes.

These distinctions should be remembered, and, in order to impress them upon your memory, I shall recapitulate.

Pachymeningitis is an inflammation of the dura only, and seldom, if ever, idiopathic (that is, produced without any apparent cause), being generally the result of an injury or disease of the cranial bones.

Idiopathic meningitis, or *leptomeningitis*, is an inflammation of both the pia and the arachnoid, of which I shall soon treat.

Tubercular meningitis is a most dangerous malady, and usually attacks children above two years of age, principally those of a strumous diathesis. It may almost be called an incurable disease; for, notwithstanding a few cases of its happy termination are on record, an incorrect diagnosis in these cases is not at all improbable.

Cerebro-spinal meningitis, according to some authors, should not strictly be classed among the nervous diseases, since it is claimed to be a result of blood-poisoning, and that it is only a form of essential fever, the inflammatory products of which are in a measure directed to the nervous system.

ACUTE IDIOPATHIC MENINGITIS (LEPTOMENINGITIS).

This is a disease which may occur at any period of life, attacking adults as well as children, being always a grave and dangerous affection. It is sometimes with difficulty differentiated from other nervous complications. In children over two years of age the meningitis is apt to be of the tubercular variety; a fact of great clinical importance, because the patient sometimes recovers from simple, but almost never from tubercular, meningitis. Hence the diagnosis of the latter will be the death-knell of your patient, bringing dismay to the agonized mother as she tremblingly awaits your gloomy verdict.

It is my desire to enable you to fully recognize acute meningitis without embarrassing your memory with lengthy and burdensome

details. What you must know are the ordinary and prominent symptoms.

The initiatory phenomenon is a chill in the adult, and a convulsion in the child (due to the preternatural mobility of its spinal over its cerebral system), as is usually the case in all acute inflammatory affections. After this, reaction will take place, and the febrile exacerbation will be more or less intense. The temperature does not run so high, however, as in many other fevers, seldom rising above 102° or 103° F. ; neither does it present any marked intermissions, but is continuous, differing in this regard from tubercular meningitis, which, like malaria, is accompanied by fever of a remittent type. It occasionally happens that these last two diseases complicate each other.

After a chill and fever, the next symptom found in simple meningitis is *headache*, localized in the frontal region or generally diffused,—not of an ordinary character, but a violent, distressing, unmistakable headache, resulting in persistent insomnia, and torturing the patient by day and by night. His screams and entreaties are beyond expression painful to hear.

The symptoms do not necessarily follow the order of sequence which I have given ; you need not, therefore, be embarrassed in your diagnosis on hearing that, instead of the chill preceding the fever, headache, etc., the symptoms have all manifested themselves at about the same time.

A characteristic *delirium* follows the headache, almost perhaps without exception, even in elderly persons, who have experienced little or no fever. Its peculiarity is, that it does not partake of the loquacious, good-natured character of *delirium tremens*, but is so violent and furious that the patient is with difficulty kept in bed, and in his rage often endeavors to injure himself. Some patients have actually succeeded in destroying themselves when carelessly watched.

Another characteristic symptom of meningitis is *vomiting*, whose peculiarities may sometimes give you a clue to the diagnosis.

A great difference will be observed between the vomiting which accompanies gastric, hepatic, or abdominal disorders, and the vomiting in meningitis. How, for instance, does vomiting take place in bilious attacks, in gastritis, hepatitis, etc. ? There is usually nausea, and the vomiting itself is painful and accompanied with

retching or straining; the patient is greatly distressed, throwing up all his food by violent spasmodic efforts. By pressure of the finger upon the epigastrium the pain will be increased and vomiting aggravated.

This phenomenon in brain-diseases offers very distinctive features; the vomiting occurring without the slightest effort,—almost *spontaneously*, in fact,—and unaccompanied by pain or nausea, features stamping it characteristically as sympathetic with disease of the brain. The vomiting resulting from gastric affections is generally relieved by the application of a mustard plaster over the epigastrium. In cerebral trouble, however, the plaster should be placed upon the nape of the neck.

I must not neglect to mention another symptom, which is *constipation*, the bowels ordinarily being quite difficult to move.

There are few or no premonitory symptoms; the onset of the disease is characteristically *sudden* and violent.

The delirium may be sometimes constant, and sometimes, though very rarely, remittent in form; at other times it is quite maniacal in character.

Convulsions seldom occur early, except in very young children and in the septic and purulent varieties of the disease.

Muscular rigidity and a tendency to opisthotonos are rare, being more frequently encountered in tubercular meningitis.

Paresis, sometimes unilateral in form, is complete or lasting only in rare cases.

When the meningeal inflammation affects more particularly the neighborhood of the motor tract,—namely, the central convolutions and paracentral lobule,—hemiplegia preceded by unilateral convulsions not infrequently occurs.

Paralysis of the cranial nerves occurs very rarely, and is more characteristically a symptom of tubercular or chronic basilar meningitis. In some few instances slight ptosis and strabismus are observable.

In *old* persons delirium and somnolence usher in the disease.

Vertigo of the cerebral type, as described in the previous lecture, frequently occurs at the onset, but is rarely a constant symptom.

Pain, constant and severe, is nearly always present in all forms of meningitis, especially in the *early* stages.

Hyperæsthesiæ of the nerves of special sense, usually diffused, appear early.

Retraction of the head, as stated above, does not often complicate acute meningitis of the convexity, as it does so frequently tubercular and cerebro-spinal meningitis.

Optic neuritis is sometimes found, especially about the end of the second week, but is observed far oftener in tubercular meningitis; it is caused by an extension of the inflammation down the sheath of the nerves, and often into the deep ocular tissues.

The sphincters are often involved towards the termination of the disease, retention of urine existing, and sometimes incontinence of fæces.

The pupils are contracted in the early stages of the disease, and dilated towards its termination; inequality of the pupils is not as common in this variety as in some other forms of meningitis.

Sloughs and bed-sores may be developed, and should be carefully guarded against.

Respiration is sometimes quickened, disturbed, and more or less impeded, but does not partake of the Cheyne-Stokes alteration of rhythm, a symptom almost pathognomonic of tubercular meningitis.

The fever in acute idiopathic meningitis is rarely high, ranging from 101° to 103° F.; it is continuous in character, with variations, but without the marked remissions of tubercular meningitis. In the purulent form the temperature may rise to 105° F. In fatal cases the temperature prior to death may rise to 106° or 108° F.

We have now considered the principal symptoms of importance characterizing the first stage of the disease. You should remember them, since they are not always described in this order. You will find these symptoms far from corresponding accurately with the typical descriptions. In fact, you need not expect the first, second, and third stages of some writers to succeed one another in regular turn.

It is only an arbitrary plan thus to divide some maladies, which custom I have followed in order to make matters clearer to your minds. Consequently, be prepared to find upon your first visit perhaps some characteristics of nearly every stage of the disease existing at the same time.

In affections of the brain, symptoms of irritation usually appear first, and are succeeded by those of depression, though this order is not absolute, the symptoms of depression being sometimes initiatory, as, for instance, in a patient stricken down by an attack of cerebral hemorrhage,—where you will have a comatose state from the commencement. In proportion to the intensity of the attack will you find the sequence of the symptoms tardy or rapid. When extremely violent, as in the *méningite foudroyante*, those of depression set in at once : you find your patient comatose on the first visit, and dead on the second. But in milder cases the different stages are better marked, symptoms of irritation often existing for several days before those of depression or coma. And this latter condition is always to be apprehended ; for it has usually a fatal termination. In these cases, the delirium passes into stupor ; you shake your patient to no avail ; you cannot arouse him ; the stupor deepens into coma,—his last sleep.

The corresponding symptoms keep pace with these changes ; and *pari passu* the pulse becomes frequent, feeble, irregular, and jerking, the skin is dry and parched, the breathing stertorous, and the patient's vital powers rapidly give way. He dies of the same coma that we find in cerebral hemorrhage, in epilepsy, in uræmia, etc., except that in epilepsy it is transitory. This coma is the second stage of meningitis, and is an *adynamic* coma. I use the word *adynamic* here as we use the term *typhoid* to express the low ebb of the vital powers, with dry tongue and feeble pulse, found in typhoid fever, but which we apply also to similar conditions present in other diseases.

Watson graphically describes the manner of dying by apnoea, asthenia, anæmia, and coma. In meningitis, the patient dies of coma. If you bear in mind all the points that have been already given, you will have little difficulty in forming a clear idea of meningitis.

You will perhaps ask, How, with this grouping of symptoms, will it be possible for us to distinguish meningitis from other diseases of the brain ? It is possible for any man to make mistakes in this as in other diseases ; but the common blunders which you would be liable to make if not forewarned I will endeavor to guard you against.

The most important point, in my estimation, is the diagnosis.

A disease which you might take to be simple meningitis, and which has misled some good diagnosticians, is—

Pericarditis.—In this disease there are sometimes indications similar to those of the foregoing affections,—viz., the head-symptoms. Should you make a hurried examination, you may conclude that the case is one of meningitis, and you will resort to active treatment,—purging, bleeding, etc.,—a treatment not quite in accordance with the modern mode of management of pericardial inflammation. I advise you to guard against this fallacy, and in such cases always to examine the thorax.

I once had a patient in whom several nervous symptoms presented themselves,—headache, vomiting, etc.,—and I was almost inclined to believe it to be a case of meningitis; for although the patient had pains in the præcordial region he referred the severest pain to the head. Certain peculiar disturbances of the pulse and of the respiration, however, soon put me on the proper track to diagnose pericarditis.

Pneumonia is another malady which sometimes presents head-symptoms similar to those of meningitis, the auscultatory evidence in the first stages of the former disease being frequently unsatisfactory. I have seen a patient who presented several suspicious symptoms of pneumonia, but I was unable to detect a crepitant râle, and there was no dulness on percussion. I finally examined the axillary space, when a distinct crepitation was manifest.

When you are at a loss to decide whether the disease is a meningitis, a pericarditis, or a pneumonia, always recollect that delirium usually accompanies a consolidation in the *apex* of the lungs, and that auscultation will furnish the desired information. Experience has taught me that the less medication employed in pneumonia the better, since it is a strictly cyclical ailment, whilst in meningitis active treatment, on the contrary, may be of some benefit.

Another source of error arises in some cases of *rheumatism*, where the blood-poison is expending its virulence upon the brain, or perhaps where symptoms formerly attributed to metastasis supervene. I remember having suddenly lost a patient once with rheumatism accompanied with suddenly-developed brain-symptoms like those of meningitis.

Examinations of the brain, in parallel cases of rheumatism, have been made by Trousseau, without discovering any confirmatory

evidence of cerebral inflammation, where death was preceded by well-marked meningeal symptoms. The efforts of other investigators, however, have been attended with contrary results. In rheumatism with an existing tendency towards the development of such complications, it is best to be ever upon your guard ; never be over-confident in your prognosis. Da Costa reports cases, moreover, of undoubted meningeal rheumatism.

Another set of head-symptoms intimately connected with the progress of *typhus* and *typhoid fevers* may also be mistaken for those of meningitis.

Typhus or "ship" fever is a disease only exceptionally seen as far west as St. Louis. In *typhoid* fever, especially in very nervous persons, the *head-symptoms* are quite prominent, and there is much nervous irritation. The Germans call this disease "Nervenfieber." To treat this fever actively would be a most unfortunate and unpardonable blunder ; therefore, examine the patient's abdomen, to ascertain whether or not it is tympanitic and painful, if pressure gives rise to gurgling and localized tenderness in the right iliac fossa, and the presence or absence of the characteristic lenticular eruption of typhoid fever.

Syphilis also is said sometimes to produce marked symptoms of cerebral and meningeal irritation ;* and still another disease is *hydrocephaloid*. Of this I had intended to speak more at length in this connection, but shall defer it until reviewing the tubercular form of meningitis.

The *prognosis* in idiopathic meningitis is very unfavorable ; so be cautious in giving your opinion, for the contingencies are such as to invest the result with great uncertainty.

The causes are excessive mental fatigue, over-exposure to the rays of the sun, severe blows on the cranium, rheumatism, and, perhaps, syphilis. Septicæmia often causes meningitis, which is then of the *purulent* variety.

De Cazal records a case of meningitis consecutive to facial erysipelas. The infection extended from a phlegmonous abscess at the inner angle of the eye, along the sheath of the optic nerve, to the base of the brain. B. Langrau reports three cases of meningitis developing under circumstances which led him to believe that

* See lecture on syphilitic nervous affections, vol. ii.

educational pressure and resulting brain-overwork constituted the exciting cause.*

The exanthemata, typhoid fever, pneumonia, and endocarditis are sometimes followed by meningitis.

ANATOMICAL APPEARANCES.

As regards the morbid appearances of this disease, recollect that the dura mater is not involved, and that the condition of the pia and of the arachnoid depends upon the intensity of the attack. There is an exudation of pus, and more or less thickening of the membranes, which present a pearly and opaque appearance. The purulence occurs sometimes very early. I have read of cases in children where it was discerned within twenty-four hours from the commencement of the disease.

The anatomical changes may involve all portions of the pia and arachnoid, but they are usually more prominent at the *convexity* than at the base. There is intense congestion of the pia. Great opacity of both the pia and arachnoid are manifest, with collections of white, yellow, and almost purulent lymph, and large quantities of serum effused under the arachnoid.

The inflammation sometimes extends to the substance of the brain and to the inner surface of the dura, which may be thickened and congested. In some instances one or two—in exceptional cases all three—membranes may be adherent. Sometimes the pia cannot be separated from the cortex without lacerating it.

The cortex cerebri may be likewise inflamed. The cerebro-spinal fluid is often increased in quantity, and cloudy or opaque. The ependyma of the ventricles may be inflamed, and the peripheral portion of the brain may be softened or present signs of marked inflammatory changes. The lateral ventricles are often distended.

There may be a gelatiniform exudation and the formation of false membranes.

The meningeal membranes in purulent meningitis are covered with greenish-yellow and sometimes offensive pus, more particularly observable at the convexity.

Charcot justly claims that it is rare to encounter purulent collections in the arachnoidean cavity; more frequently pus is found

* Gray, *Annual of the Universal Medical Sciences*, Sajous, 1891.

under the arachnoid membrane; sometimes, however, the pus collects between the inner table of the skull and the dura; finally, under the influence produced by cranial hyperostosis, pus accumulates between the two layers of the dura.

It is an important clinical fact that in very young children the disease is not ushered in by a chill, but by convulsions,—a result partially due to their liability to convulsions in consequence, as West says, of the preternatural mobility of their nervous system, which is therefore easily irritated by trifling disturbances, whether originating from teething, worms, fevers, or the exanthemata.

As to *treatment*, I have not much to say. I do not believe in the efficacy of tartar emetic, calomel, or repeated and copious venesections in the treatment of inflammation, having known iritis to occur during mercurial ptyalism. You can accomplish something by administering drastic purgatives, and I have great faith in the good effects produced by cathartics, as derivatives, in certain affections of the brain. As a matter of course, they must be used with caution, or the result will be unfavorable; but there is a medium in all things, even in the administration of beef tea or water. At the very beginning of the disease, a purgative is highly beneficial,—the old “ten and ten” (calomel, ten grains, jalap, ten grains) being probably as good as anything else you can use; sometimes croton oil or elaterium.

Cold applications to the head, by means of ice-bags, must not be neglected; *but never fail to give precautionary directions about applying the ice at intervals, and moving it about; for I have seen cases where the scalp had been actually frozen.* Ergot is of advantage in meningitis, by its action upon the vaso-motor nerves, causing contraction of the blood-vessels; and its action may be intensified by the addition of bromide of potassium. Iodide of potassium also bears a good reputation, being with many writers the remedy *par excellence*; it is to be given freely and boldly, especially where a rheumatic or a syphilitic taint exists.

Should the disease become subacute, shave the scalp, and pustulate it with croton oil: this measure, in children especially, frequently has an admirable effect. In cases with sthenic symptoms, you might resort to venesection, or to the application of leeches to the mastoid processes, the anus, the Schneiderian membrane, or the occipital protuberance. Cupping, also, is very useful. After the

cups use counter-irritants; apply mustard plasters to the feet, wrap them in warm fomentations and cover them with blankets. Remember Boerhaave's celebrated maxim: "Keep the head cool, the feet warm, the bowels regular." In this, as in other inflammatory fevers, give judicious nourishment, like beef tea, milk, or other food easy of digestion and absorption; for in most of these troubles there is a moment of crisis which is liable to carry off the patient unless the vital powers have been sufficiently supported. If, in spite of all this, your patient should show signs of approaching death, you might then even venture to administer stimulants.

LECTURE VI.

TUBERCULAR MENINGITIS.

Acute Idiopathic Meningitis—Anatomical Lesions a Peculiarity—Acute Hydrocephalus
—Symptoms: Period of Invasion, Gradual Impairment of Health, Change of Habits and Temper, Headache—Importance of Cephalalgia—Stages: Slow Pulse, Suspicious Respiration, Cerebral Maculae, Boat-shaped Abdomen, Flush and Pallor, Cephalic Cry, Remission in Fever, Increased Somnolence, Coma, Changes in Paralytic Phenomena—Sources of Error: Bilious Intermittent Fever, Typhoid Fever, Hydrocephaloid of Marshall Hall, Partial Anæmia—Optic Neuritis—Prognosis—Treatment—Koch's Injections in Tubercular Meningitis.

GENTLEMEN,—In my last lecture, when speaking of *acute idiopathic meningitis*, I remarked that one of its most salient and characteristic features is its occurrence in children generally under the age of two years or above the age of ten, or in adults. I also stated that in children acute meningitis commences with convulsions, and that their early appearance or recurrence is quite characteristic of this disease. Simple meningitis we found to be an inflammatory affection, accompanied by fever, and hence readily discriminated from active hyperæmia of the brain, by placing the thermometer in the axilla. *Convulsions* preceding acute disease in children are simply premonitory *symptomatic* indications of greater or less trouble, *but are significant of no particular pathological state*. They are produced by the presence of worms, dentition, the exanthemata, epilepsy, brain-affections, or toxæmia; in short, they occur in all cases of acute affections or where some reflex irritation has produced central or functional nervous disturbances. If you have carefully retained what I told you when describing simple meningitis, you will recollect that I also referred digressively to tubercular meningitis, stating that it usually attacks children *between the ages of two and ten years*. It is a disease peculiar to childhood, and is generally treated of in works upon the diseases of children, but it may occasionally attack adults of

a scrofulous diathesis, or in whom tuberculous deposits exist in the lungs, mesentery, or other organs.

The anatomical lesions of this malady differ from those of simple meningitis. The membranes involved in both diseases are the same, it is true, but the latter affects the convexity, while tubercular meningitis attacks the *base*, of the brain, whence the French derive their name of "*méningite de la base*." You occasionally find described a disease called *acute hydrocephalus*. In what respects does it essentially differ from simple acute idiopathic, or from tubercular, meningitis? It is only another name for tubercular meningitis. It is called acute hydrocephalus because the disease was so termed by Dr. Whytt, of Edinburgh, who originally described it as "acute dropsy of the brain," "*ventricular meningitis*." This name does not, however, express the original character of the malady itself, but only one of its results. Moreover, in all forms of tubercular meningitis there is usually an exudation of fluid into the ventricles, causing softening by pressure, and a kind of saponified, friable, œdematous condition of the surrounding brain-structure, while in simple meningitis these cavities are, as a rule, empty. The name acute hydrocephalus, therefore, expresses only a *result*; and you might with as much propriety call typhoid fever a disease of Peyer's glands, though the ulceration of these follicles is but a consequence of the action of its peculiar *materies morbi*. Now, one of the principal lesions in tubercular meningitis is this dropsical effusion in the ventricles of the brain; but we have others just as important, as, for instance, the tuberculous deposit at the base of the brain, or in the meshes of the pia mater, or in the fissure of Sylvius. Acute hydrocephalus is, therefore, an improper appellation.

Perhaps it would not be unsuitable in this place for me to say that, in my opinion, it is inadvisable to designate a disease by the name of an individual. "Duchenne's disease" implies nothing in relation to its pathology or symptomatology; but "progressive locomotor ataxia," or "sclerosis of the posterior columns of the spinal cord" (Hammond), immediately conveys to the mind the principal characteristic features of the disorder. The same may be said in regard to "Bright's disease" for certain affections of the kidneys, and "Addison's disease" for certain lesions of the suprarenal capsules. Acute hydrocephalus is the older, and

tubercular meningitis the more recent, designation for the same disorder.

What are the principal symptoms of tubercular meningitis? I say *principal*, because I will not consider its minutiae. With Trousseau, I believe that too great importance cannot be attached to the *period of invasion* as a diagnostic point. A due recognition of this is an important preliminary step in untying the Gordian knot of diagnosis. In acute idiopathic meningitis there is a *stormy* outset, the fever and brain-symptoms rendering the period of invasion strikingly alarming. But in tubercular meningitis the period of invasion is so very peculiar and insidious that it cannot ordinarily be mistaken by any careful observer. The child, being of strumous diathesis, of scrofulous parentage or ancestry, ceases to be healthy. It is not at once seized with tubercular meningitis while enjoying perfect health, as in acute meningitis, but there is a *gradual failing of the general health*, and emaciation commences. This change, the beginning of which was scarcely noticed, at last becomes not only evident but striking. It is sometimes rapid, and the child, who perhaps was formerly fat and vigorous, the very picture of health, soon loses flesh, and becomes greatly emaciated. While this is in progress, another condition attracts our notice,—one on which Trousseau lays great stress. This is the *moral* change, manifested in the *habits and temper* of the child. The little patient, who had been formerly perhaps of playful disposition, grows irritable and peevish; objects which formerly were incitements to playfulness now cease to attract attention. The child becomes irritable, taciturn, morose, and disagreeable, indifferent to pettings and caressings, which formerly were eagerly sought for and fully appreciated, and experiences no interest or pleasure in the company of its little playfellows or in its accustomed frolics. At length the mother becomes seriously alarmed, and is positive in asserting that there is something the matter with the child, who very likely at this period begins to evince the existence of *violent headache*. This cephalalgia will come on paroxysmally, and the child, whilst running about, will suddenly stop, lean against something, put its hand to its head, and complain of violent pain. It will also be seized with *vomiting*,—not the ordinary vomiting from gastric disturbances, but true *cerebral vomiting*, which I described in my last lecture.

Of course the parents are now extremely solicitous, and a physician is sent for. Well may they be frightened; for although headache is not infrequent in children, still, when it follows slow emaciation and the other symptoms described, the mother feels that there is probably some grave affection undermining the health of the little one, and her suspicions are well founded. If the physician summoned be the regular family attendant, he has undoubtedly already observed the nature and suspected the cause of the ominous changes in the child, and his case is pretty well made out before he arrives at the house. But if he be a stranger unacquainted with the family, he will learn, upon inquiring into the history of the case, that the child is suffering from severe headache, perhaps vomiting, and, moreover, has been ailing for an indefinite period, with well-marked change of character and moral perversion, its health meanwhile gradually declining. Do not lose sight of these facts; they are exceedingly important; you must recollect them, as they will prove serviceable. In order to show the importance of *cephalgia* in children as a symptom, I will relate a case which occurred in my own experience.

I was once called upon to visit a young child suffering from severe headache. The attending physician was absent, and my immediate presence was desired. On my way to the house, it occurred to me that there might be something grave about this case, requiring, as it did, the immediate attendance of a medical man to relieve the suffering, not even awaiting the return of the regular doctor. On examination, I found that the headache had lasted several days, that contraction of the pupils existed, with photophobia, intolerance of sound, and marked fever, the temperature being 103° F. I immediately jumped at a diagnosis, which I caution you never to do; for the disease might have been remittent fever, which in children often produces violent head-symptoms, which are cured by quinine. But I felt justified in the conclusion I had so rapidly arrived at, because, upon inquiring concerning the previous health of the patient, I learned that it had failed gradually, that the attack had been insidious, and, as the mother expressed it, "she didn't exactly know when the child first got sick." After hearing this, reviewing the other symptoms, and revolving them in my mind, I was convinced; and, to my sorrow, I diagnosticated tubercular meningitis, without com-

municating my fears to the family. The same afternoon I met the attending physician, informed him of my visit to the child, and communicated to him my fears and diagnosis. He asserted that I was mistaken,—that the child was only suffering from a “*neuralgic*” affection. I reminded him that Dr. West states that neuralgia is of exceedingly rare occurrence in children ; nor would it be accompanied by fever, or preceded by slow and gradual deterioration of health. We parted, and a few days afterwards I was informed that my diagnosis had been correct, and that death had rapidly supervened. When you consider that Trousseau, with all his opportunities, immense practice, and enormous consultation experience, emphatically states that he met with only *two* cases of tubercular meningitis that recovered, and that even then it was highly probable that he had been mistaken in his diagnosis, you will readily be convinced of the hopelessness of the affection. Therefore, *beware of neuralgia* in children ; for, generally, there is something more serious underlying the superficial symptoms.

Let us now consider the stages of this illness. I have already stated that I do not in general approve of an arbitrary division of disease into stages ; as they so seldom correspond to the real accession and development of the morbid phenomena, which vary in different cases, untrammelled by artificial limitations. It is a source of fallacy, therefore ; but, as it is the custom to divide disease into stages, I shall give you the different symptoms which, as nearly as possible, constitute those of tubercular meningitis. We have first the period of invasion, already described, with irritability, fretfulness, moral changes, etc. ; then fever, accompanied by a slightly accelerated pulse, delirium, headache, cerebral vomiting, restlessness, wakefulness, constipation, and *contraction of the pupils*. These characterize what is termed the *first stage* of the disease.

We have here the symptoms of excitation, of hyperæmia, with the addition of fever. When this stage has passed (always supposing that we have this regular succession of phenomena), the *pulse* becomes remarkably *slow*,—the so-called “*cerebral pulse*,”—falling to seventy, sixty, or even fifty per minute in very young children, and there exists likewise an interference with its rhythm. A peculiar *embarrassment of respiration*, not enough to amount to a dyspnoea or an orthopnoea, is produced ; but there is a *rhyth-*

mical disturbance which sometimes, though not always, corresponds to the change in the pulse. About this time a kind of mottling appears upon the skin, which has been described as *cerebral maculae*, and upon drawing your finger over it, quite distinct traces are left, which fade gradually. This is not, however, essentially characteristic of the disease.

You will observe a *peculiar appearance of the abdomen*, constituting a sort of *boat-shaped* excavation; the intestines sink, the abdominal parietes shrink, and the superior spinous processes of the ilia come out in bold relief. This symptom, though not pathognomonic, is still quite characteristic of the affection, and should always be sought.

There are remarkable alternations of *flushing and pallor* of the countenance present in all the varieties of meningitis, due perhaps to a peculiar state of the vaso-motor nerves, causing the alternate contraction and relaxation of the blood-vessels which they control; and when you examine the child, you will sometimes observe its face glowing and congested, and there may be a sudden accession of marked wanness. From these symptoms you will justly conclude that you are dealing with a case of meningitis, and that the danger is imminent and threatening.

There is also developed a *cephalic cry*, the result of pain,—a peculiar, piercing, wild, automatic shriek, never to be forgotten when once heard. After you have seen a few cases of tubercular meningitis, these different symptoms will become indelibly impressed upon your memory. The headache, fever, flushing, pallor, sunken abdomen, stupor, disturbances of pulse and respiration, are quite characteristic, but, in reality, are merely the prodromes of the third stage,—*coma*.

In the second stage there are very often *remissions of the fever*, sometimes so marked that they may lead you to think you have made a mistake in diagnosis, and you will administer quinine, hoping to check the fever, as probably of malarial origin. But remember that this is merely clutching at a straw, as these remissions are peculiarly characteristic of the disease, and may be observed even in its last stage, after coma has set in.

During the second stage a marked symptom is *increased somnolence*. In the first stage, owing to its great irritability, the child is annoyed by the doctor's presence, lights, sounds, etc.; but later

it is sleepy, stupid, lethargic; the hyperæsthesia having disappeared, you may pinch the child without its manifesting much pain or annoyance. After observing the delirium, maculæ, cephalic cry, headache, boat-shaped abdomen, flushings, peculiarities of pulse and respiration, and the remissions in the fever, you may imagine that there is no possible source of error. Bear in mind, as I have already told you, that it is not always possible to make a diagnosis at first. You need not commit yourself by giving a positive opinion, but should endeavor, by proper observation, to arrive at correct conclusions, even if it take several days to do so. Also, keep well in mind the fact that these signs are common to different diseases, and do not attach too much importance to any isolated symptom. Judge only by the combination of manifestations, sequence of events, and concurrence of conditions, or you will be led into error.

The *third* stage is *coma*, as in simple meningitis. The pulse is exceedingly feeble and frequent, too frequent to be counted. There is perspiration and a clammy feeling of the skin, the *pupils* are *dilated*, and we have the symptoms of depression, with their ordinary paralytic phenomena. Hammond says, "In young infants there is a reduction of the temperature of the body below the normal standard, which lasts throughout the whole of this period." Rogers regarded this reduction, preceded as it is by a higher temperature and followed during the succeeding stage by another elevation, as "pathognomonic of tubercular meningitis."

These paralytic phenomena have this peculiarity, that they are inclined to change, so that we may first have a paresis or a hemiplegia; strabismus and ptosis may occur; or first one group of muscles will be paralyzed, to be followed by the paralysis of another, and then again the paralysis may entirely pass away. Occasionally there are convulsions, absent at other times; but if they are present, it is always towards the *termination* of the disease, while in *acute* idiopathic meningitis they always occur at the *outset*. The coma finally becomes more and more hopeless and intense, the pulse more frequent and feeble, the skin more clammy and moist, the urine and *fæces* are involuntarily evacuated, and death ends the scene.

SPECIAL SYMPTOMS OF TUBERCULAR MENINGITIS.

Having described the *general* symptoms of this disease, I will now refer more particularly to its *special* symptoms.

The premonitory symptoms of the period of *invasion*, which, as before described, while *progressive* is yet extraordinarily *gradual* and insidious, present a previous history of ill health and malaise; the child complaining of numerous ill-defined disorders concurrently with more or less emaciation, at first not strikingly evident, yet eventually unmistakable.

Associated with this is a *moral* change, *sui generis*, on which too great stress cannot be laid. It is a change in the habits, inclinations, and temper of the child. Upon this point we cannot too emphatically claim attention, as a diagnostic link in the chain of sequences. The changes in the *moral* and in the *physical* conditions progress *pari passu*.

The *headache* often exists several weeks before the onset of the acute symptoms, which may be due to the deposit of tubercles in the pia and arachnoid, a condition believed to exist prior to the *inflammatory* changes of these last-mentioned membranes.

Headache is almost invariably present, and is a peculiarly distressing symptom, continuous in the main, but at times qualified by more or less marked remissions and exacerbations. The "hydrocephalic cry" is *probably* the result of intense pain.

Gowers says, "In *rare* cases meningitis runs its course with little or no pain. This is especially rare in tubercular inflammation, but not uncommon in the secondary *purulent* meningitis of *septicæmia* and in the simple meningitis of some other blood-states."*

Convulsions occur in the last stages of tubercular meningitis, and very seldom at the onset, as in acute idiopathic meningitis. They are usually associated with septic processes, and may be unilateral or bilateral.

Rigidity of the muscles of the neck and a tendency to opisthotonos are more frequently met with in tubercular and cerebro-spinal meningitis than in any other of its varieties.

Paresis and hemiplegia become more particularly manifest in the

* Italics my own.

last week of the disease, appearing and disappearing, declining and intensifying, as is usual with symptoms characterized by *mutability*, a quality essentially pertaining to the paralytic phenomena of tubercular meningitis, and probably dependent upon variations in the cerebral circulation. As stated before, the symptoms of kinesodic origin are more prominently developed when the inflammation is more highly pronounced in the vicinity of the central convolutions and paracentral lobule.

Paralysis of the cranial nerves gives rise to frequent symptomatic manifestations of tubercular meningitis, particularly ptosis, strabismus, and facial palsy, inequality of the pupil, and optic neuritis. These symptoms are most frequently encountered at the end of the first week.

Towards the end of the second week *delirium* and *lethargy* deepen into coma, and muscular rigidity is increased; the cranial-nerve disturbances are correspondingly intensified, and so are the local convulsions and local paralysees.

Disturbances of the *æsthesodic* zone are rarely marked, except, perhaps, as hyperæsthesiæ.

Aphasia is often present in tubercular meningitis, which is not remarkable when we consider the dominant intensity of the inflammatory changes and morbid disturbances in the fissure of Sylvius, where the deposit and consequent irritation of tubercles are greatest. The facial palsy is not very considerable, consisting principally of *inequality of the labial commissures*, with more or less inactivity of the orbicularis palpebrarum,—a condition not to be confounded with *partial* ptosis from paralysis of the levator palpebræ, due to palsy of the third pair. In all brain diseases *the palpebral openings* should be carefully examined and compared.

The aphasia is ataxic or motor in character, but may be associated also with the amnesic variety.

Gowers asserts that "*inequality of pupil* is often present when other ocular symptoms (including neuritis) are absent."

The *pulse* at the commencement of the malady is increased in frequency and correspondingly diminished in volume; during the second week it becomes cerebral in character, being often reduced to sixty, fifty, or forty per minute. Towards the termination of the disease it may vary from one hundred and thirty to one hundred and eighty per minute, or may be uncountable.

The temperature in the beginning is elevated three or four degrees, or even more, as time passes, sometimes reaching 105° or 106° F. In some cases it falls *below* the *normal* shortly before death. In *purulent* meningitis it has been known to rise as high as 106° or 108° F. It has been asserted that in some exceptionally rare cases the disease runs its course *without fever*.

Gowers quotes (from Bokai) that, "in one recorded case, on the seventeenth day of the disease, the day before death, the temperature was only 93° F."

Optic neuritis is often observable about the end of the first week.

Towards the end of the disease, the sphincters often become relaxed, the skin cold and clammy, and the eyes glazed and not infrequently covered with a muco-purulent secretion.

The auditory nerve, in consequence of its intimate intracranial association with the facial, is sometimes affected.

The hypoglossal nerve is rarely affected; if it is, the tongue will deviate somewhat on protrusion.

Sloughs and bed-sores sometimes occur.

The boat-shaped abdomen, alternations of flushings and pallor, remissions in the fever, "hydrocephalic cry," cerebral maculæ, and disturbances of the rhythm of the pulse and respiration, so characteristic, have been sufficiently described, the former frequently corresponding with the changes in the latter.

Embarrassment of the respiration is a pathognomonic symptom of tubercular meningitis; it is a rhythmical disturbance of the respiratory act, and corresponds really to a "Cheyne-Stokes" respiration.

ANATOMICAL APPEARANCES OF TUBERCULAR MENINGITIS.

Post-mortem examination reveals the presence of inflammation of the membranes,—namely, the pia and the arachnoid. In addition, tubercular granulations of a miliary character are scattered here and there. In the early stages they are very small and quite transparent. They are principally seated in the pia, and can be felt as well as seen. In many places they are aggregated in colonies. They afterwards increase in size, and become semi-opaque in appearance. The inflammation of the membranes is more developed at the *base* of the brain, especially in the neigh-

borhood of the fissures of Sylvius and the optic chiasma. There is frequently an increase of cerebro-spinal fluid, particularly at the *base*.

There is often a copious exudation of lymph, almost semi-consistent, in some instances more or less mingled with pus.

As stated before, there is frequently ventricular dropsy, with an inflammatory thickening of the ependyma, accompanied with more or less softening in the vicinity.

The granulations are composed of lymphoid cells. These bodies are usually distributed in the perivascular spaces. There is often an optic neuritis by extension of the inflammation along the sheaths of the optic nerves.

Some dispute has arisen among eminent authorities as to whether the gray gelatinous granulations found in tubercular meningitis are tubercles or not. Valleix, Rilliet and Barthez, Barrier, Grisolle, Meigs and Pepper, and others, as quoted by Hammond, regard them as such. Grisolle expresses himself clearly on this point. "We have no doubt," he says, "that these granulations are tubercles in a rudimentary state; for we have many times, in the same subject, followed the morbid product in its different phases of evolution from the amorphous condition to the fully-developed tubercle."

Hammond, again, quotes Robin and Bouchut as believing in their non-tubercular nature, in which opinion he does not sustain them.

Dr. Whytt, of Edinburgh, was probably the first writer (1768) who published systematic observations on this subject, under the caption of "Hydrocephalus Internus."

It is conceded by most writers that the deposit of tubercles is a condition prior to the development of meningeal inflammation.

It is also generally admitted that tubercles in these cases are usually found in other organs, particularly in the lungs and the mesentery.

The dropsical effusion in the ventricles, in some instances, distends *all* the ventricles.

"The indications of occlusion of the communication between the fourth ventricle and the surface, already mentioned, are more frequent in tubercular than in simple meningitis." (Gowers.)

The same authority states that "tubercles may be found in the membranes when there is no sign of inflammation, in cases of general tuberculosis, and they may be accompanied by symptoms of cerebral disturbance resembling those caused by inflammation."

In cases of tubercular meningitis the bacilli of tubercle are present.

While the weight of authority is opposed to the injection of Koch's lymph for general diagnostic purposes, in consequence of the dangerous reactions that follow, we are inclined to believe that tubercular meningitis forms an exception in this respect, because of the jeopardy in which the patient's life is placed, and that objections of this kind would be more than counterbalanced by the results and chances of these injections.

"It is claimed that the descending horn of the lateral ventricle is particularly liable to become over-distended in tubercular meningitis. The exudation into the ventricles may be purulent in rare instances. As a rule, it consists of serous fluid which is more or less turbid from an admixture of white blood-corpuscles and epithelium. Tubercles may be often detected in the ependyma, and along the vessels of the choroid plexus." (Ranney.)

The morbid anatomy of tubercular meningitis may be divided as follows: (1) tubercular infiltration; (2) an inflammatory exudation in the meshes of the pia; (3) ventricular dropsy.

The favorite site of the miliary tubercular deposits in this disease is at the *base* of the brain; they are most frequently encountered at the bifurcations of the blood-vessels, in the neighborhood of the Sylvian fissures and of the optic chiasma, and in the region of the circle of Willis.

Gowers and Ranney have frequently observed miliary deposits in the neighborhood of the longitudinal fissure.

According to the *Mercredi Médical*, M. Jean Charcot and M. Souques, from investigations in a number of cases of phthisis where the brain had become secondarily involved, have found that the commonest seat of attack of the tubercular process is the *paracentral lobule*. The authors are of the opinion that this is due to the peculiar circulatory arrangements in and around this region.*

* New York Medical Journal, August 1, 1891.

DIAGNOSIS.

What are the diseases with which tubercular meningitis may be confounded? I may mention, first, *bilious remittent fever*. This we can readily diagnosticate by giving quinine, a specific for this disease. If you are doubtful of your diagnosis, do not hesitate to administer this drug freely and boldly; for in cerebral affections it is best to give it in large and *sedative* doses, in preference to smaller ones, which only irritate and produce no compensating beneficial effects.

Sometimes it becomes difficult to distinguish tubercular meningitis from *typhoid fever*, because the symptoms of the latter, especially those of a cerebral character, often bear a great similarity to those of the former disease. In children the enteric symptoms—diarrhœa and tympanites—are often absent, while the *brain-symptoms* are very prominent. But here you must remember the pathognomonic symptom, which is the peculiar disturbance in the respiration, always present in tubercular meningitis, never in typhoid fever. It is important to diagnosticate correctly in this respect, as typhoid fever is not often fatal in children, while tubercular meningitis is always an “*incurable affection*.”

There is another condition to be considered in this connection, the *hydrocephaloid* of Marshall Hall. This is but another name applied to general anæmia of the brain, causing cerebral symptoms, which is developed in children after protracted exhaustive diseases, especially bowel-complaints lasting for a long time. It is very often the result of cholera infantum. In this affection, when the diarrhœa exists, the first thing in order is to check its progress. We should give brandy, and even ammonia, if the system will bear it; but we should inevitably produce disastrous and fatal consequences if we were to mistake it for congestion or inflammation and treat accordingly these hydrocephaloid symptoms, which bear a striking analogy to the phenomena presented during the course of the meningeal affections we have just studied. On the contrary, gentlemen, it is under such circumstances that you must energetically sustain the vital powers. In this manner a cure may be effected; while, if these measures be neglected, death will surely follow. This proves the validity of an assertion made in a former lecture, of the similarity of the symptoms of

cerebral anæmia and cerebral hyperæmia. In all these forms of meningitis there is an anæmia of the capillary vessels, produced towards the termination of the malady. The pathological law which I have so often given you can be once more applied in these affections,—I mean the state of depression which follows the primary one of irritation. In complete anæmia the abolition of the functions is absolute, and only the symptoms of depression are evident. But in partial anæmia we have the symptoms of excitation preceding those of depression; and this is exactly what happens in meningitis.

In conclusion, allow me to state that the prognosis is of the utmost gravity: death is the usual termination.

As to treatment, there is none which has ever been successful. Notwithstanding this, you should spare no efforts to save the life of your patient; and I therefore recommend to you, as indorsed by the highest authorities, the iodide of potassium, freely administered in combination with the bromide of potassium. Use counter-irritants, and pustulate the scalp with croton oil. Do not lose sight of the hygienic treatment. Tonics, beef tea, etc., must never be neglected. In the last stages you may give stimulants. During the prodromic stage cod-liver oil and the hypophosphites should be administered in all cases.

Regarding ventricular surgery in tubercular meningitis, I quote from a recent able article by Dr. L. Bremer, of St. Louis, "An Outline of Cerebral Surgery," *St. Louis Medical Review*, October, 1891: "Puncture of the ventricles for *acute* hydrocephalus is also looked upon by most brain-surgeons with disfavor, because, it is claimed, it represents only a localization of miliary tuberculosis. But whether there are not cases of acute tubercular hydrocephalus which might be saved by timely surgical interference from almost absolutely certain death, is still an open question. If we bear in mind the surprising effect of the opening of the abdomen in tubercular peritonitis, and, furthermore, that crops of miliary tubercles have been observed to appear and disappear in the larynx, we must look upon miliary tuberculosis as being curable, perhaps more curable than any other form of the tubercular processes, and we certainly have no right yet to condemn an operation for a *meningitis* which is done on the same principle as that for a *peritonitis*."

LECTURE VII.

CEREBRO-SPINAL MENINGITIS.

Anatomical Characters—Its Nature—An Essential Fever—Malignant Scarlet Fever—Malarial Fevers—Three Forms: Simple, Fulminant, and Purpuric—Clinical History—First Form—Symptoms—Brain, Spinal, and General: Chills and Fever, Vomiting, Pain, Decubitus—Second Form—Third Form—Reabsorbent Fever—Death from Asthenia or Coma—Generalities—Pathological Anatomy—Prognosis—Treatment—Hygiene—Morphine for Rachialgia—Iodide and Bromide of Potassium, Fluid Extract of Ergot, Belladonna, Quinine, Salicylate and Benzoate of Sodium.

GENTLEMEN,—In to-night's lecture I propose to treat of an interesting affection, generally prevailing epidemically, and very rarely sporadically,—an exceedingly fatal disease, with which physicians find it difficult to contend. Those who fully appreciate its nature and are candid will readily admit that they dread its occurrence and have little or no control over its progress. This disease is cerebro-spinal meningitis, sometimes denominated "spotted fever," and known by many other names,—the consideration of which need not detain us.

Cerebro-spinal meningitis is an *inflammation of the membranes of the brain and of the spinal cord*. Hence the distinction is easy between this affection and affections involving only the membranes of the brain. It is really a cerebral meningitis *plus* a spinal meningitis, the brain and the spinal cord being both enveloped by the same membranes.

As regards its nature, several conflicting opinions exist. Some contend that it is essentially a disease of the nervous system; others, that it is malarial in origin; and still others, that it belongs to the *essential* fevers. To discuss this question fully would require much time, and, after all, perhaps we should not arrive at a satisfactory conclusion. I do not think it a disease of the nervous system, any more than I think that typhoid fever should be ranked under such a classification. I believe, with most neuro-patholo-

gists, that it is the result of a blood-poison,—actually an essential fever, whose whole force and violence are expended upon the nervous system, not unlike the *materies morbi* existing in typhoid fever, which attacks Peyer's glands, causing prominence of the abdominal symptoms, because the principal outburst of the storm is spent upon those organs. Hence, as we do not know the actual character of typhoid fever, it might be better, with Dr. Wood, to call it an enteric fever. It is highly probable that just as a particular *materies morbi* produces enteric fever, so does the blood-poison in cerebro-spinal meningitis induce inflammation of the cerebro-spinal coverings.

Cerebro-spinal meningitis has been held by some to be merely a variety of malignant scarlet fever. Such a theory scarcely needs refutation; almost all authors agree that cerebro-spinal meningitis possesses nothing in common with the exanthematous or eruptive fevers. The reason for my not believing it to have any relationship to malarial fever is, that quinine, which possesses such remarkably specific powers in, and is the best and most reliable of all antidotes to, malarial toxæmia, is utterly powerless to arrest or even influence its course. I have given it myself, in large and bold doses, and, notwithstanding my firm belief in its efficacy and extraordinary powers in most febrile complaints, I must confess its total failure in my hands to control this disease in the slightest degree; and almost all other medicines with which I have tried to check the devastations of this dreadful scourge have likewise produced little or no satisfactory result. In reference to the question of its being a kind of "cerebral typhus," I can safely assert that cerebro-spinal meningitis has, in my opinion, no relationship whatever to typhus. There may be, it is true, a similarity between some of the clinical phenomena of both diseases; yet in reviewing its history we must come to the conclusion that cerebro-spinal meningitis is *not* a form of typhus fever. Typhus is a very contagious disease,—far more so than cerebro-spinal meningitis. Neither is typhus a disease of such short duration, nor does it uniformly involve the cerebro-spinal meninges. In the cerebro-spinal form of meningitis there are marked symptoms of spinal origin, although there are also purely intracranial ones; but the former are never present in typhus. Neither is there any similarity between the eruptions of the two diseases. Cerebro-

spinal meningitis is, consequently, in all probability a disease *sui generis*, and should be grouped with the essential fevers.

You will perhaps ask yourselves, If this be the case, why speak of it in connection with diseases of the nervous system? I simply do so as a matter of convenience and custom. Some of its pathognomonic symptoms are entirely referable to the nervous system. I think it advisable, therefore, to speak of it at the present moment, in order to differentiate the disease from other forms of meningitis; my chief idea being to give you a proper conception of its peculiarities.

We must next consider the principal symptoms of this affection. As regards their classification, I believe, with Russell Reynolds, that there are three forms or varieties of cerebro-spinal meningitis. I shall not describe them at length, but shall merely review their principal features. The first is the *simple* or *ordinary* variety; the second, the *fulminant*; and the third, the *purpuric*.

The ordinary variety, like the others, rarely occurs sporadically, the disease being generally *epidemic*, and often devastating whole communities. It occurs mainly in crowded, unhealthy, ill-ventilated places, or where hygienic requirements are neglected. This often happens in prisons, camps, workhouses, and hospitals. There is perhaps a short period of invasion, marked by general malaise, and, in abortive cases, by headache and constipation; but I believe the disease rarely aborts, and the patient but too often dies. The mortality is always greatest during the early part of an epidemic. This is usually the rule under such circumstances, the greatest malignancy generally occurring in the earlier cases, and thus spending itself; and, no matter how great your care or how unceasing and earnest your efforts, they will at this period usually prove futile. During its prevalence you will be on the alert: as during epidemic visitations of variola, when everybody grows alarmed at the appearance of no matter what kind of an eruption, and backache, or as in scarlatina, when the slightest throat-trouble causes the greatest anxiety, so during the existence in the community of cerebro-spinal meningitis the least pain in the occipital region gives occasion for the most serious apprehension.

The phenomena of the *simple form* of cerebro-spinal meningitis are first *chill*, which is followed by *fever*; then more or less *vomit-*

ing, accompanied by violent *pain* in the *head*, *nucha*, and *back*, and *delirium*.

In order to recognize an acquaintance, we ordinarily endeavor to recall the peculiar appearance and general conformation of his features, not the particular form or character of any single lineament of his countenance, but the special combination which characterizes his physiognomy. So it is in disease, the peculiarities of which are not always represented by one symptom, but by a concurrence of many, and also by their peculiar grouping and arrangement. In cerebro-spinal meningitis, therefore, we consider, first, the *epidemic prevalence of the disease*, the *initiatory chill and fever*, the *vomiting*, the *delirium*, the *pain in the nape of the neck* and in the *spinal region*, and the *general cutaneous hyperæsthesia*.

Vomiting is always a marked, persistent, and obstinate symptom. The pain in the nucha is violent and enduring. One of the important peculiarities of the clinical phenomena is, that the pain in the spinal region is intensely severe. It is lancinating, —darting to the four extremities of the body, its violence being greatly aggravated by the least movement; and hence the decubitus of the patient is very peculiar, and sometimes causes the meningitis to be mistaken for rheumatism, especially as the hyperæsthesia may be misinterpreted.

In cerebro-spinal meningitis we have brain-symptoms, the meninges of the brain being involved; and we therefore find the violent pain in the head, with the existence of prominent delirium. Among the other brain-symptoms are vomiting, insomnia, constipation, and contraction of the pupils, which afterwards dilate. The *spinal* symptoms are the lancinating lumbar and sacral pains, aggravated by every movement, and accompanied by more or less spasm, mostly of the muscles of the neck, thus fixing the head backwards on the spine. In some cases the spasmodic contraction is so extensive as to draw the whole body violently and firmly backwards, producing *opisthotonos*: usually the spasm is limited to the nuchal region.

The *general* symptoms are fever, with a temperature varying from 103° – 4° to 106° – 7° F., more or less constipation, sometimes diarrhoea, anorexia, prostration of the vital powers, due to the implication of the nervous centres, and insomnia, or, in some cases, stupor. There is often an herpetic eruption in the neighborhood

of the lips, and ecchymoses on the body, giving the disease the name of "spotted fever." These symptoms constitute the ordinary form of cerebro-spinal meningitis. Of course they vary with different epidemics; and so at times the eruption will be greater or less, or even absent; the disease also differing in violence, intensity, and duration.

The *second* is the fulminant type. This means the "thundering" form, and in truth it is also a very lethal type of the disease. Its malignity is so great that it has proved fatal in five hours. If a blood-poison is of sufficient virulence to produce such terrible results, what can a physician accomplish in his efforts to oppose its progress? He can achieve nothing. What constitute the peculiarities of this form? Have you ever seen the stage of collapse of epidemic cholera, the algid state, as it is called? If you have, you will remember the ghastly pallor, the shrunk condition and clammy coldness of the skin, the pulselessness and general prostration, the cyanotic appearance of the mucous membranes, and the rapid sinking of the vital powers. These phenomena are also seen in the fulminant form of cerebro-spinal meningitis, with apoplectic phenomena in addition. You thus have the appearances of the algid state of cholera, *plus* coma. There is little possibility of reaction, and at your first visit your patient is often moribund,—at the second, dead.

The *third* form is the purpuric. This form occurs as a combination either of the first and the second, or of the first with purpuric symptoms, in consequence of extensive and profound blood-poisoning, the result of dyscrasia or true necræmia. There is a tendency to extravasation of blood in the subcutaneous and submucous tissues; the capillaries, friable from want of tonicity, rupture; hemorrhage follows, and petechiæ appear. No matter what combination of symptoms accompany this variety, there is an intense, malignant blood-poisoning, a necræmia, and a conjoint appearance of spots and blotches of a purpuric hue upon the surface of the body.

There is another remarkable fact in regard to the symptomatology of this disease, referred to by Ziemssen. Patients who do not die during the first or second week of the attack are not entirely free from danger. Very often a fever is developed, called the "reabsorbent fever," which is a pyæmic condition, induced by the absorption of peccant matter. In this disease, as well as

in simple cerebral meningitis, a sero-purulent exudation collects in the subarachnoidean spaces, and at the base of the brain, causing an impairment in the functions of the nerves of special sense.

After the primary fever has disappeared, the reabsorbent fever develops during convalescence. An absorption of the exudations occurs; and if the resulting pyæmia be not fatal, the patient evinces signs of a commencing protracted recovery, though still having one more danger to incur,—that of a marasmus, not unlike *tabes mesenterica*. The nervous centres presiding over the functions of nutrition are probably at fault, damaged by the violence of the acute stage; a pernicious diarrhœa and progressive wasting soon reduce the patient, in spite of beef tea, wine, and tonics, to a mere skeleton. Finally, death by asthenia occurs; although at earlier periods of the disease it may be from coma.

While we have considered the nature and etiology of this malady, some little remains to be said of its pathology or pathological anatomy. I believe that cerebro-spinal meningitis is primarily a disease of the blood, with inflammation of the pia and arachnoid of the cerebrum and medulla spinalis, resulting from toxæmia, and attended by an effusion of serum, lymph, and pus. If death is produced by *neeræmia*, no trace of meningeal inflammation is found, *because there has not been sufficient time for the inflammatory condition to produce the transudation.*

In view of what has been said, you may readily infer the prognosis: it is of very grave import, and experienced physicians fear to encounter this dread disease. I hesitate to consider its treatment, having tried almost everything with but slight beneficial results. I have lost many cases, some dying in spite of every effort to save them. Others recover almost unaccountably. Of course, on reference to your books you will find modes of treatment detailed *ad infinitum*; but after testing their efficiency at the bedside and observing the results, your faith in medication will probably be almost completely shattered. Try for yourselves, and profit by your own experience. I doubt if we shall ever be able to make great progress in mastering epidemics. In all cases, be guided by your experience, your convictions, and your earnest desire to achieve your utmost. I do not wish to trammel your memories with a catalogue of therapeutical resources, as I have found no satisfactory result from any medication in the cases which

have fallen under my observation. Keep up nutrition and sustain the vital powers. During rachialgia you may use morphine hypodermically, taking care not to give too large a dose. You might administer iodide and bromide of potassium and fluid extract of ergot; the two latter, by acting on the vaso-motor nerves, may control the hyperæmia of the meninges. Belladonna, cannabis Indica, quinine salicylate and benzoate of sodium, counter-irritants, etc., have all been recommended. I have only to add in regard to remedies that their number is generally in direct proportion to the hopelessness of the affection. Their multiplicity corresponds with their inefficiency.

GENERALITIES.

Cerebro-spinal meningitis has especially prevailed in the United States and in Europe, notably in Sweden, Germany, France, and Ireland. It is a disease of all ages; in some epidemics children have been more frequently attacked. It is more prevalent in the first two decades of life. Winter and spring are the periods during which its epidemic influence is most felt. Though very probably an *infectious* disease, it is in no sense *contagious*, nor does one attack afford protection from another.

As in all acute affections, a rigor or chill may constitute an initiatory symptom. Vomiting and headache are very prominent symptoms after the chill, and are usually accompanied with intense rachialgia. The headache is always very severe, and often constant. The hyperæsthesiæ of the skin and of the nerves of special sense are very acute. Delirium and headache are concomitant. The rachialgia is accompanied by erratic pains in various muscles, is increased by movement, and may radiate in various directions, especially towards the loins and limbs. Rigidity of the muscles of the neck and back and retraction of the head are eminently characteristic features. Movement or flexion of the neck is extremely painful. Trismus has been noticed in a few cases. Convulsions are noticed at times, but are not limited to any particular stage of the disease. The abdomen is sometimes retracted, and the legs may be drawn up.

“Cerebro-spinal fever may during an epidemic complicate other acute maladies, and mix its symptoms curiously with them. With an attack of this disease the trouble does not pass off, for it may

leave behind it all kinds of want of power and local palsies, besides derangements of vision, *permanent deafness*, impaired intelligence, epilepsy, persistent headache, chronic meningitis, which may be its cause, and chronic hydrocephalus." (Da Costa.)

The blood-changes are very rapid and intense in cerebro-spinal meningitis. The necræmia, the conspicuous head-symptoms, and the peculiar eruptions are features of this affection comparable with those of typhus fever. In cerebro-spinal fever, however, the blood-deterioration is more intensified and more quickly accomplished. The delirium is less, the fever is not so high, and the duration of the disease is shorter.

Da Costa says, "In an autopsy on a child that died in twenty-four hours, I found the blood diffuent and black; in an adult patient who had been sick but two days, I detected blowing sounds in the heart, evidently of blood-origin. The poisoned blood unquestionably gives rise to many of the nervous symptoms, and it is on the blood and the nervous centres that the poison mainly acts."

The temperature and pulse vary much: the former may rise from 104° to 106° F.; the latter, from one hundred and twenty to one hundred and fifty beats to the minute.

In view of the skin-eruptions, the disease in this country has sometimes been called "*spotted fever*." Purpura, herpes, erythema, and urticaria are frequently encountered.

Paralysis of the cranial nerves, deviation of the eyes to one side, inequality of the pupils, and strabismus are not infrequent. Conjunctivitis, optic neuritis, and ulceration of the cornea may be observed. As in acute idiopathic meningitis, the pupils are primarily contracted and in the later stages dilated.

Permanent deafness and blindness are not infrequent among the sequelæ.

In character and duration the disease presents great variations. Gowers states that "in the most acute cases the patient quickly becomes comatose, and dies at the end of one or two days, sometimes even in five or six hours from the onset." The acute form has been termed *fulminant*. On the other hand, mild cases sometimes occur, where the symptoms are headache, pain in the back, and slight rigidity of the neck-muscles,—a form that has been termed, somewhat inaptly, *abortive*.

The mortality varies in different epidemics: it is sometimes very high.

Pneumonia and bronchitis are occasional complications. Arthritis is sometimes another complication, and should not be confounded with rheumatism, "as the head-symptoms, the state of the muscles of the neck, and the dissimilar course of the malady soon clear up the diagnosis." (Da Costa.)

The disease has sometimes been confounded with scarlatina. "*An extremely rapid pulse* * would be in favor of the view of the case being scarlatina." (Da Costa.)

In some cases there is persistent vomiting.

The characteristic eruption is described as follows by Da Costa: "The cutaneous surface is frequently spotted with a red eruption, erythematous and roseolous,—an eruption which often becomes brownish and then for the most part rapidly petechial, and which is wholly uninfluenced by pressure; or the purple spots may be seen from the start."

Ziemssen states "that the high temperatures are often interrupted by long-continued normal temperatures."

Lung-troubles may predominate much more in some epidemics than in others.

"Uræmia may cause muscular rigidity, convulsions, and coma, thus occasionally giving rise to symptoms somewhat like those of cerebro-spinal meningitis (Murchison); but the temperature is normal, and other symptoms of each malady are usually recognizable. It must be remembered that in children retraction of the head may occur from rheumatic affection of the muscles and other causes." (Gowers.)

"A considerable diagnostic difficulty is presented by cases in which the meningitis runs an almost latent course. It is sometimes found, after death, in cases of pneumonia in which it was not suspected during life, the headache and delirium having been ascribed to the pulmonary malady. Unequivocal symptoms of cerebral mischief, however slight they may be, should always receive attention in this disease.

"General hyperæsthesia sometimes first suggests the presence of more than the lung-disease." (Gowers.)

* Italics my own.

ANATOMICAL APPEARANCES.

There is intense opacity and congestion of the pia of the brain and cord, and sometimes the latter also is inflamed. The ventricles of the brain are sometimes distended with fluid, or even with pus. The ependyma is frequently involved in the inflammatory changes. The brain contains foci of softening, purulent collections, and minute hemorrhages. Exudations of lymph, with dépôts of pus, are not infrequently found in the membranes, which are often thickened and in places adherent. The microscope sometimes shows lymphoid cells along the course of the vessels.

"The proved dependence of many acute specific diseases on micro-organisms has suggested the probability that epidemic meningitis is due to a similar cause." (Ziemssen.)

BACTERIOLOGY OF EPIDEMIC CEREBRO-SPINAL MENINGITIS.

"Several interesting studies of this subject from a bacteriological stand-point have been recently made, of which that by Adenot is perhaps the most exhaustive. His conclusions are: 1. Several varieties of microbes have already been found in the meningeal exudates. 2. The kinds thus far found in meningitis are: *a*, the pneumococcus; *b*, the streptococcus pyogenes; *c*, the intra-cellular micrococcus of Weichselbaum; *d*, the probable bacillus of typhoid fever; *e*, the probable staphylococcus pyogenes; *f*, the pneumobacillus of Friedländer; *g*, undetermined microbes. 3. It is probable that further research will enable us to enlarge this number. All forms of true meningitis are probably of microbic origin. 4. Meningitis is primary and secondary. The last-named form of the disease, developed in the course of infectious diseases, is often the result of the mixed infection, and due to a different microbe from that which excited the primary infection. 5. Certain microbes can locate primarily in the meninges, which do not, however, choose that location habitually. We have reason to believe this of the typhoid bacillus. We may thus admit a cerebral typhoid without typhoid fever. 6. The microbes find their way to the meninges either through a direct route, as in otitis, or by way of the circulation. The last-named course is far the more frequent.

"Huguenin, from an extended study of the sources of infection

in meningitis, reaches the conclusion that purulent meningitis is always due to micro-organisms, but that serous meningitis and fibrinous meningitis are due to a cause which is not yet determined. He recognizes five forms of bacilli capable of exciting meningitis, adding to those mentioned by Adenot the bacillus meningitis of Neumann and Scheffer.

"Bonome claims to have isolated an encapsulated diplostreptococcus from a meningeal exudate of epidemic cerebro-spinal meningitis, which he regards as different from all other forms yet found in this disease. He does not consider them as a new family of bacteria, but as, perhaps, only a variety of the lancet-like diplococcus of Fraenkel. Netter reports a case of suppurative meningitis following a pistol-shot in the mouth, in which he found the pneumococcus associated with the staphylococcus pyogenes aureus. The author considers the case as confirmatory of experiments which he had made in 1886, producing meningitis by trephining and introducing a culture of pneumococci under the dura, the pistol-ball in the man's case laying bare the under surface of the brain, just as the trephine had a superior surface in the animals experimented upon."*

* Landon Carter Gray, *Annual of the Universal Medical Sciences*, Sajous, 1891.

AUTHORS TABULAR STATEMENT
Of the Differential Diagnosis of Acute Leptomeningitis, Tubercular Meningitis, Cerebro-Spinal Meningitis, Chronic Vertical Meningitis, and Chronic Basilar Meningitis.

| LEPTOMENINGITIS. | TUBERCULAR MENINGITIS. | CEREBRO-SPINAL MENINGITIS. | CHRONIC VERTICAL MENINGITIS. | CHRONIC BASILAR MENINGITIS. |
|---|--|---|--|---|
| No period of invasion. Onset stormy, sudden. Period of occurrence, any age. Initiated by chill in adults, by convulsions in children. Temperature, 102°-104° F. Fever of continuous type. Headache violent, excessive, continuous (particularly in early stages). Insomnia persistent. Cerebral vomiting. Constipation. Delirium constant, noisy, and sometimes maniacal. Convulsions rare in adults, save in septic and purulent forms. Muscular rigidity and opisthotonos rare. Paralysis rare; partial or complete, dependent upon pathological implication of cortico-spinal tracts; more pronounced when paracentral lobule is involved; paresis sometimes unilateral. Hemiplegia preceded by unilateral convulsions sometimes occurs. Paralysis of the cranial nerves rare. In old persons, delirium ushers in the disease. | Period of invasion marked and often characteristic. Onset gradual, slow, insidious. Age, generally from two to ten years. Convulsions, particularly in later stages. Temperature in beginning from 103° to 104° F., in some rare cases subnormal. Fever of remittent type. Headache violent but remittent. Insomnia persistent in early stages. Cerebral vomiting. Constipation. Delirium. Muscular rigidities and opisthotonos not infrequent. Paralysis may be partial or complete, particularly towards later stages; inclination to mutability of symptoms of paralysis. Paralysis of the cranial nerves frequent. Cerebral vertigo may exist. Hyperesthesia in earlier stages replaced by anesthesia in later stages. Optic neuritis often present at end of first week. Sphincters relaxed towards end of disease. | <i>First or Ordinary Form.</i> Generally epidemic in character, followed by fever. Vomiting. Headache. Pain in nucha and back. Delirium. General cutaneous hyperesthesia. Spinal pain severe, lancinating and darting. Insomnia. Constipation usually. Pupils at first contracted, later dilated. Opisthotonos. Temperature, 103°-104° F. Prostration of vital powers. Coma. Herpetic eruption in neighborhood of lips. Eclymoses of body. <i>Second or Fulminating Type.</i> Frequently fatal. General prostration. Cyanotic appearance of skin and mucous membranes. Coma or general apoplectic phenomena. <i>Third or Purpura Type.</i> Symptoms the result of a profound blood-poisoning. | Headache diffused and persistent. Intensity not so great as in the more acute form. Lethargy towards termination. Weakness of limbs. Vertigo. Sphincters more or less affected (late in disease). Articulation at times impaired. Spasms of special groups of muscles. Mental faculties often dulled. Convulsions at times. Cranial nerves are not so frequently involved as in some other forms,—especially basilar. Disturbances of sensation,—especially neuralgic pains. Paresis more predominant than paralysis. Optic neuritis rare. General health falls in some cases, but in many instances is unaffected. Constipation. Cerebral vomiting. Coma. Duration several months to several years. Intermitence of symptoms. | Headache early, persistent, and a most prominent symptom. Epileptiform attacks frequent, concomitant with the cephalalgia. Convulsive movements of a limb, muscle, or set of muscles unattended with loss of consciousness. Tonic spasms of muscles of one or more of the extremities, especially those of the arms; neck and head may be similarly affected; head in consequence held in an abnormal position. Individual muscles of the face not usually involved. Paralysis sooner or later. Facial palsy. Difficulty in articulation. Some or many of the motor nerves of the eye may be involved. Unilateral paralysis of the third nerve common, especially in syphilitic cases. Dilatation of the pupils. Vertigo. Mental confusion. Aphasia. Anisopsia, limited or dif- |

DIFFERENTIAL DIAGNOSIS OF THE VARIETIES OF MENINGITIS.—(Continued.)

| LEPTOMENINGITIS. | TUBERCULAR MENINGITIS. | CEREBRO-SPINAL MENINGITIS. | CHRONIC VERTICAL MENINGITIS. | CHRONIC BASILAR MENINGITIS. |
|--|--|--|--|---|
| <p>Cerebral vertigo at onset, rarely constant.</p> <p>Hyperaesthesia of nerves of special sense; photophobia; trinitus aurium.</p> <p>Optic neuritis sometimes present, especially towards end of second week.</p> <p>Sphincters paralyzed towards termination.</p> <p>Inequality of pupils not so common as in many other forms; contracted at first, and afterwards dilated.</p> <p>Bed-sores sometimes developed and sometimes quickened and disturbed.</p> <p>Temperature in septic and purulent forms 102°-106° F. or higher.</p> <p>Pulse feeble, irregular, and jerky.</p> <p>Skin dry and parched.</p> <p>Coma in final stage.</p> | <p>Inequality of pupils may occur.</p> <p>Bed-sores.</p> <p>Respiration shows disturbances of rhythm (similar to Cheyne-Stokes).</p> <p>Coma in last stage.</p> <p>Moral and physical changes as prodromes.</p> <p>Cerebral maculae.</p> <p>Scaphoid abdomen.</p> <p>Alterations of flushing and pallor.</p> <p>Hydrocephalic cry.</p> <p>Skin clammy, pupils dilated, in last stage.</p> <p>Strabismus and ptosis frequent.</p> <p>Rogers regards a reduction of temperature preceded by a higher temperature, and followed in a succeeding stage by another elevation, as pathognomonic of tubercular meningitis.</p> <p>Aphasia often present.</p> <p>Facial palsy,—not generally marked.</p> <p>Pulse towards termination of second stage becomes slow and cerebral in character.</p> <p>Sometimes ulceration of cornea, eyes glazed, and at times covered by a mucopurulent secretion.</p> <p>Auditory nerve may be affected.</p> <p>Hypoglossal nerve rarely involved.</p> | <p><i>Third or Purpuric Type.</i></p> <p>Tendencies to subcutaneous and submucous extravasations of blood.</p> <p>"Reabsorbent fever."</p> <p>Impairment of nerves of special sense.</p> <p>Marasmus.</p> <p>Pernicious diarrhoea.</p> <p>Diseases of all ages more prevalent in the first and second decades.</p> <p>Infectious, not contagious.</p> <p>Delirium and headache common.</p> <p>Trismus rare.</p> <p>Convulsions when present not limited to any particular stage.</p> <p>Abdomen at times retracted and legs drawn up.</p> <p>Impairment of vision, hearing, and intelligence <i>often permanent.</i></p> <p>Epilepsy and persistent headache sometimes follow convalescence.</p> <p>Skin eruptions, — purpura, herpes, erythema, urticaria, petechiae.</p> <p>Paralysis of the cranial nerves.</p> <p>Inequality of pupils.</p> <p>Strabismus not infrequent.</p> <p>Conjunctivitis sometimes present.</p> <p>Optic neuritis.</p> <p>Ulceration of cornea may occur.</p> <p>Pupils at first contracted, later dilated as in leptomeningitis.</p> | <p>Symptomatology not so marked when compared with severity and extent of lesions.</p> | <p>Disturbances of vision frequent; often appear early; may be general or special.</p> <p>Disturbances of the cranial nerves common.</p> <p>Optic neuritis often present.</p> <p>Vision sometimes seriously impaired.</p> <p>Hearing at times lost.</p> <p>Mind, as a rule, not affected to a notable degree.</p> <p>Mutability of symptoms may be present.</p> <p>No set of symptoms that may not at times be absent; no pathognomonic symptom of chronic meningitis.</p> <p>In ordinary pyrexia, headache ceases when delirium begins.</p> <p>In meningitis, headache continues and coexists with delirium.</p> |

LECTURE VIII.

PACHYMENINGITIS.

Forms of Meningitis—Pachymeningitis—Pachymeningitis Externa—Pachymeningitis Interna or Hæmorrhagica—Etiology: Blows, Injuries, Ozæna, Otorrhœa—Anatomical Characters—Clinical History—Hæmatoma of the Dura Mater—Inflammation of the Cerebral Sinuses—Thrombosis—Metastatic Abscesses in the Lungs—Symptoms—Causes of Death—Treatment—Prognosis—Pathological Anatomy—Cranial Surgery in Pachymeningitis.

GENTLEMEN,—In my last lecture, while speaking to you upon the subject of cerebro-spinal meningitis, I discussed its connection with different diseases, such as typhus fever, scarlatina, malarial fever, etc., with which, at least by some authors, it has been confounded. I compared its clinical and pathological phenomena with those of each of these diseases, affirming my belief that it was not in any manner connected with them. I moreover took the position that it is not *primarily* a nervous affection, but an *essential fever*, somewhat resembling typhus in its action, and that in cerebro-spinal meningitis a peculiar *materies morbi* probably exists, whose action upon the cerebro-spinal nervous system causes the inflammation of the meninges.

In the consideration of the different diseases of the membranes covering the brain, we have thus far reviewed: first, acute idiopathic; secondly, tuberculous; thirdly, cerebro-spinal, meningitis. In each of these diseases there is, as we have already seen, an inflammation of the pia mater as well as of the arachnoid. We now come to the description of the fourth form, the last but one that we will study. It is a variety of rare occurrence, against which, however, you must be constantly on your guard, never allowing it to elude your vigilance, as it is apt to deceive the inexperienced physician. This affection is known as *pachymeningitis*, or inflammation of the dura mater.

If you have carefully followed me in my previous lectures, you will recollect that in the other varieties of meningitis the dura

is not involved. In the present malady, however, the inflammation is almost exclusively limited to that membrane, the others remaining healthy.

The dura mater is composed of two layers,—an inner one, having an epithelial surface, and an outer one, thicker and serving as a periosteum.

Pachymeningitis is distinguished as *external* when the outer layer of the dura is inflamed, and *internal* when the inner dural membrane is involved. The latter form is known as hæmatoma of the dura mater.

EXTERNAL PACHYMENINGITIS.

This affection is generally secondary. Fractures of the cranial bones produce it sometimes, by an extravasation of blood between the bone and the dura. Erysipelas has been known to be a factor in its production.

It should be distinctly remembered that in pachymeningitis the dura alone is affected.

Gowers very appropriately states that, “of the three membranes that enclose the brain, only two are pathologically separable, since the arachnoid and pia mater always suffer together. The separate inflammation of the dura mater, ‘*pachymeningitis*,’ is much less common than the affection of the pia-arachnoid, which is commonly meant when ‘*meningitis*’ is spoken of. The affection of the soft membranes has been of late termed ‘*leptomeningitis*,’ in more precise antithesis to pachymeningitis.”

Caries of the petrous and ethmoid bones and of the upper cervical vertebræ are well-known causes of pachymeningitis.

“From my experience, which is not entirely exhausted by the preceding cases, I cannot hold *idiopathic* pachymeningitis, independent of external injury or syphilis, to be so rare a disease as authors affirm. I believe the disease is frequently mistaken, and supposed to be a *febris larvata*, on account of the regular intermissions, or more frequently a *cephalæa rheumatica*.

“At first sight it may appear strange that this inflammation is distinguished by such intense painfulness. It must be remembered, however, that the dura mater cerebri consists of two layers, of which the outer forms the periosteum with which the dura mater proper is coherent. The great painfulness in consequence

of inflammation is possessed by the dura mater in common with the periosteum of other bones. The dura mater of the vertebral canal, separated from the periosteum, is, according to my experience, far less painful in inflammation than the dura mater cerebri. Also degenerations, ossifications, and even inflammation of the falx cerebri appeared, in a few cases which have occurred to me, not to pursue a very painful course. In the vertebral canal an isolated inflammation of the dura mater occurs indeed only seldom, and on that account we have no perfectly pure observations. However, I have not observed the pains occurring here in such severity, although perhaps they proceeded from other parts. If the disease takes a more chronic course, through which the dura mater unites almost inseparably with the skull, then the severe pains do not always occur. Thus was it with the seventy-two-year-old man, where the skull could not be separated from the dura mater, and where, nevertheless, no headache had been present.*

"The intermittence is also peculiar; it often occurs as distinctly periodic as in intermittent fever, but mostly manifests itself irregularly, so that rather long complete intermissions are distinguished. Here, again, we recognize the correspondence of the dura mater with the periosteum of other parts. In periostitis generally the pain comes on more severely during the night, or it has even longer intermissions. Other authors also mention the intermittence of the symptoms of the disease. Especially many observations of the kind are found in the works of the distinguished Lallemand. (*Recherches sur l'Encéphale.*) . . .

"But in very acute cases these intermissions appear to be absent, or perhaps they were not observed in the beginning of the disease, before medical treatment was commenced."†

Pachymeningitis is however, we believe, rarely idiopathic, being almost always dependent upon some secondary cause; hence, when it exists, we can generally suspect the nature of its etiology. It

* "Probably the strong coherence of the periosteum to the bones, and the great tension in consequence of inflammatory swelling, through which an injurious and painful pressure on the nerves arises, contribute much to the painfulness. At least the periostitis which always occurs after fractures or after amputations is not very painful, in case the torn edges of the periosteum do not sustain any tension and are not exposed to pressure."

† Schroeder van der Kolk, *Pathology and Therapeutics of Mental Diseases.*

differs considerably in this respect from acute idiopathic meningitis ; a child, for instance, is often seized with the latter affection without our being in the slightest degree able to ascertain the exciting cause, but in pachymeningitis there are certain generally-recognized influences leading to its production. The most ordinary of these are *severe blows upon the head*, and *external violence, fractures or fissures in the skull*, to which we may add diseases of the *bones of the cranium*, such as *caries* (syphilitic or otherwise), resulting from *ozæna*.

Another very common and important source of pachymeningitis, which I wish you always to recollect, and one which is not sufficiently appreciated or recognized, is otorrhœa, with caries of the temporal bones. Otorrhœa is a frequent sequel to scarlatina or other exanthema, such as rubeola or variola. In these diseases there is usually an affection of the throat, an inflammation of the pharynx, which is more or less persistent, situated in the mucous membrane of the fauces, which by continuity of structure may be transmitted along the mucous membrane of the Eustachian tube and finally involve the middle and internal ear. This rapidly destroys the ossicula auris, attacks the deeper layers of bone, and finally extends to the dura mater. Jaccoud states that when the disorder follows an injury, as a blow upon the head, the starting-point of the inflammation is in the membrane lining the external surface of the skull,—the pericranium. The inflammatory condition of the pericranium, for reasons not obvious, causes the inflammation of the dura mater : as there is no very evident connection between these membranes, we cannot very easily explain the mode of transmission of the inflammation ; though, after all, it might be communicated or propagated through the osseous structure.

Considering what I have said in regard to otorrhœa, you will readily understand the necessity and importance of its energetic treatment, notwithstanding the representations of parents that its cure is attended with danger. As a rule, mothers do not wish an interference with any discharge. This is an old-fashioned but still prevalent prejudice. Formerly it was considered very injudicious on the part of a physician to arrest or check purulent discharges. I have seen little children, covered with eczema, scratch, suffer, and pass sleepless nights, simply because the family physi-

cian acquiesced in the wishes of the mother, who, according to some traditional notion, imagined that brain-disease would inevitably follow the disappearance of the eruption. These ideas, as I have said already, are held not only in regard to otorrhœa, but also with respect to *cutaneous* eruptions, and have some authoritative weight in their support. Owing to such opinions, many an otorrhœa has been allowed to run its pernicious course, involving caries of the neighboring bone, inflammation of the dura, and the death of the patient. It is always well to respect the feelings of a mother, but you should never allow yourselves to be dictated to by any one governed by prejudice. Rather decline the responsibility of the case. "The fact that the inflammation," Hammond says, "sometimes alternates with skin-eruptions is interesting, and has been repeatedly noted. A case of the kind was not long since under my care. It was that of a gentleman who had attacks of acute pain in the head, accompanied with all the phenomena of paralysis of the left third nerve. There was effusion of lymph upon both optic disks, the result probably of old optic neuritis. Curiously enough, these attacks alternated with an eczematous affection involving the trunk and especially the breast. On the disappearance of the skin-disease under remedial measures, his head-symptoms immediately recurred, and, when they were relieved by the action of the iodide of potassium, he was again attacked with eczema."

In this case the intracranial affection was evidently devoid of great or proximate danger. *Not so with pachymeningitis.* Hence, as a possible cause of so grave a malady, you must at once arrest an otorrhœa; do not temporize with it for a moment when occurring under the circumstances above named; *abolish it* as speedily as you can, for, on account of the existing systemic conditions, the inflammatory action which creates the discharge is exceedingly prone to extend by continuity to the dura. Nor am I willing to relax anything of stringency or rigor in the above rule for otorrhœas of long standing, especially when they have ensued upon the exanthemata, or upon a purulent *otitis media* of whatever origin. The most chronic cases may all at once develop acute symptoms of cerebral character most insidiously and quickly carry off your patient.

In order to illustrate to you the suddenness of death in some

such cases, I will relate an incident to you which came under my direct observation. Before its recital, I can conceive that you may perhaps inquire if in pachymeningitis we do not first observe symptoms of irritation followed by those of depression, or marked headache, convulsions, vomiting, contraction of the pupils, etc., previous to the advent of coma. In answer, I would say that in some cases such symptoms may be present, but in others absent; and the first symptomatic indications will often be those of depression, those of irritation having been so slight as to have been entirely overlooked, and coma will follow. But I must relate my case, as an illustration from actual experience is always much more instructive than a long disquisition. I wish particularly to impress upon you the necessity of caution, by citing to you not my triumphs but my mistakes, in order that you may be prevented from falling into errors similar to my own.

Some years ago I was the physician of a most respected and interesting family, one of whose members was a young lady about eighteen. She was a charming girl, very intelligent and highly accomplished, and had had during childhood an attack of scarlatina, followed by an otorrhœa so obstinate and persistent as to defy all treatment. Dr. Spencer, a distinguished specialist in aural surgery, had treated her without success. About the time of the sad occurrence I am relating, the young lady was noticed to be failing in health, which was all the history I could glean. She was not very sick, but the mother had become uneasy and sent for me. I also learned that there had been some fever, and, being somewhat in a hurry, I diagnosticated on the spur of the moment *intermittent fever*, which was then quite prevalent. Upon questioning the mother further, I learned that the girl had had severe headache for a few days previously, and also that she imagined her daughter was at times somewhat delirious. This was perfectly compatible with my diagnosis. As she also had a sore throat, I proceeded to examine it carefully. Bringing her near the gaslight, I noticed that the light greatly hurt her eyes, in consequence of photophobia, but paid no attention to this important fact. Still believing that she had malarial fever, and her tongue being coated, I prescribed calomel and quinine, and then left, promising to return the next day. The mother, being nervous and anxious about her child, followed me to the

door and asked for my opinion. I immediately proceeded to reassure her, firmly believing that the quinine would do its work, so I told her that there was no cause for alarm, as the young lady would be well in a few days. About eleven o'clock the same night I received a message from a neighboring physician, who desired my presence at the house of my patient, stating also that she was dying. I thought it was probably some hysterical trouble, making him over-anxious, but still went, intending to reassure him. I had scarcely entered the room before I recognized that she was comatose, and the same minute I appreciated my sad error of diagnosis. I had overlooked the importance of the otorrhœa, although aware of its existence, which in such cases points to a contingent pachymeningitis, in the forcible language of Niemeyer, like an impending "sword of Damocles." The otorrhœa, photophobia, headache, constipation, and delirium were all known to me at my first visit, yet I overlooked the danger and gave an encouraging prognosis! I was baffled and mortified, as the lady died that same night, and I should have anticipated the unfortunate termination of her illness. It was a lesson which is still indelibly impressed upon my mind. If this mistake of mine can be at all beneficial to you, if an otorrhœa with cerebral symptoms can make you apprehensive, and sound the note of alarm, when presented for your consideration, then I am amply repaid in having related my melancholy experience. You will have remarked how very few were the symptoms of irritation in this case, how rapidly coma supervened, carrying the patient off before alarming symptoms had manifested themselves. You see, therefore, that pachymeningitis is an affection to be dreaded, and you will bear in mind the possibility of an extension of disease from the ear to the dura mater.

INFLAMMATION AND THROMBOSIS OF THE CEREBRAL SINUSES.

You are all acquainted with the peculiarities of the dura mater, its sinuses, and their peculiar anatomical relations. Now, when an inflammatory condition of the dura exists, there will be developed a tendency to the formation of thrombi in the cerebral sinuses, with subsequent inflammation of their walls. The inflammation of the dura may be propagated to the sinuses, stasis of blood will occur within them, and a clot or thrombus being

formed will interrupt the circulation and clog their cavity. This is one of the contingent dangers of inflammation of the dura mater, and according to the location of the primary cause will a particular sinus become involved. In ozæna and caries of the *ethmoid bone*, the longitudinal sinuses will be implicated, while in caries of the petrous portion of the temporal bone the lateral and petrosal sinuses will be inflamed.

From your knowledge of thrombosis and embolism, you are aware that the interference with the circulation in the cerebral sinuses is not the only danger to be apprehended, as there may be another important complication,—a metastatic abscess in the lung. You should always remember that thrombosis may result in embolism, as sometimes occurs, for instance, after inflammation of the uterine sinuses, and also in phlebitis resulting from fractures or other causes. We have already seen how the embolus becomes detached and is taken to the right ventricle and thence to the lungs, where, if large enough, it will plug up the pulmonary artery, or one of its important branches, producing death by apnœa. But if the clot be small and derived from a *suppurative focus*, a *metastatic abscess* will be produced in the lung. This is exactly what sometimes happens in pachymeningitis. Ideas upon this subject were, up to a recent date, of a very crude character. You will now be able fully to realize the danger of this disorder, and also to understand its mode of origin.

Thrombosis of the cerebral sinuses, with resulting metastatic abscesses, may be suspected when *rigors* occur during the course of pachymeningitis.

Gerhardt states that a grave suspicion of thrombosis of the transverse sinus exists when there is "less fulness of the jugular vein drawing its blood from the obstructed sinus."

This fact would be corroborated by a symptom upon which Griesinger lays stress, which, it is true, he found in only one case,—namely, "a circumscribed painful œdema behind the ear;" although, as Niemeyer observes, "in caries of the mastoid process this œdema (which Griesinger calls a *phlegmasia alba dolens* in miniature) may arise from other causes than from the extension of the thrombus though the *emissaria Santorini* which pass out in the sigmoid fossa."

ANATOMICAL APPEARANCES OF PACHYMENINGITIS.

The dura mater is thickened, and may be found attached to the cranial bone, with accompanying ossification of the proliferated connective tissues. There is great swelling, increased vascularity, and œdema of the dura.

Pus may be found between the dura and the bone ; occasionally between the two layers of the dura. The pia-arachnoid may be adherent to the internal layer of the dura.

Ecchymoses of diminutive character are observed.

The dura is at times discolored and softened.

When pus has been formed, the dura may be detached more or less extensively from the bone.

It is not easy to ascertain whether the inflammation of the sinuses is primary or secondary to the thrombosis.

The thrombi may be adherent to the walls of the sinus, or broken down and infiltrated with offensive pus.

"Along with these changes we generally find those of otitis interna and extensive caries of the petrous bone,—viz., destruction of the drum, absence of the ossicula, polypoid proliferations of the mucous membrane, the tympanum full of pus, which also infiltrates the labyrinth, cochlea, and mastoid cells." (Niemeyer.)

"The example of Macewen, in scraping out and thoroughly disinfecting the middle ear when this has become hopelessly destroyed by the primary purulent inflammation, should be more generally followed in operating for cerebral abscess traceable to this cause, as such a measure is well calculated to protect the patient against subsequent infection. The foregoing cases furnish abundant proof of the utility, as a life-saving operation, of timely surgical interference in all cases where well-defined cerebral symptoms point to the extension of the purulent process from the ear to the brain or its *meninges*, even when well-marked focal symptoms are absent,* as an exploratory operation under strict antiseptic precautions would not constitute a source of danger ; and if pus is found, early incision, drainage, and disinfection may often succeed in saving a life that under any other form of treatment would be hopelessly lost." †

* Italics my own.

† Senn, Surgery of the Brain, Ann. Univ. Med. Sci., Sajous, 1888.

LESIONS FROM EAR-DISEASE.

G. Newton Pitt, in the first of the Goulstonian lectures for 1890 upon the subject of cerebral lesions, gives an interesting analysis of fifty-seven fatal cases of ear-disease affecting the contents of the cranial cavity. Nearly all the cases occurred in patients under thirty years of age, only nine being over thirty. Four were babies less than three years old. *As an illustration of the difficulties attending a diagnosis in ear-disease as associated with brain-symptoms, he mentions the fact that in more than one-sixth of this series the patient died without any otorrhœa having been noticed.** In all the cases in which pyæmia occurred, the onset was preceded by thrombosis of the lateral sinus. Death was caused in all but two of these cases by intracranial complications,—among them abscess, mastoid suppuration, *meningitis*, and sinus thrombosis. Of the abscess cases, three were in the cerebellum, one in the pons, two in the centrum ovale, and the remaining twelve in the temporo-sphenoidal lobes. In only two of the abscess cases was there any fever due to the abscess. The temperature was rarely high with uncomplicated cerebral abscess; 2° F. above the normal in six cases. In eight it was high; *three of these had meningitis*, two thrombosis of the lateral sinus. The author reaches the following conclusions:

“First, abscess in the temporo-sphenoidal lobe, which is by far the most common situation, is often associated with an inflamed or sloughing dura mater over the anterior surface of the petrous bone or pus beneath it. Other complications are infrequent, except meningitis, generally due to extension or rupture of the abscess. The abscesses are almost always very close to the roof of the tympanum. Imperfect drainage of the (middle) ear is frequently, if not invariably, the origin of the mischief. Mastoid suppuration often affects the posterior surface of the petrous bone, but it may be associated with disease limited to the middle fossa of the skull. Cerebral abscess only occurs when the otorrhœa has lasted for months or years. The symptoms usually come on insidiously. Rigors, pyrexia, and optic neuritis are all infrequent in uncomplicated cases, but all occur occasionally. A headache of intense

* Italics my own.

severity, and a dull, sluggish mental state, are the two most characteristic symptoms. Cerebellar abscesses are less common, and will probably be associated with disease of the dura mater behind the petrous bone, or with thrombosis of the sinus.

"With regard to thrombosis of the lateral sinus occurring as a complication of ear-disease, it is stated to have occurred twenty-two times. In some of the cases there was well-marked phlebitis, but not in all. The thrombus was suppurating in more than half. The thrombosis developed in some of the cases directly from inflammation or necrosis of the petrous bone, the dura being inflamed or sloughed over it. In three cases there was a collection of pus outside. In other cases infection had spread from disease of the mastoid cells or of the posterior wall of the tympanum by means of the conveyance which emptied into the sinus, the dura mater not having been infected. The thrombus giving rise to sinus thrombosis is generally of some standing, but not always. The chief symptoms, nearly always of sudden onset, are pyrexia, rigors, pain in the occipital region and in the neck, associated with a septicæmic condition; well-marked optic neuritis may be present. The appearance of acute pulmonary mischief is almost conclusive of thrombosis. The average duration is about three weeks, and death is generally from pyæmia."*

Unfortunately, there are no particular or pathognomonic symptoms of this affection. You should, however, be constantly on your guard as to the existence of the conditions of the *primary* disturbance, which, with the history, will give you a clue to the diagnosis as well as the treatment. If the patient has received a violent blow upon the head, if he has otorrhœa or ozæna of long standing, and before death exhibits marked general cerebral symptoms, you may safely conclude that the disease is probably pachymeningitis. The symptoms of the affection may be obscure, but the etiology remains clear. Hence it is that I do not wish to dwell upon unimportant symptoms, the main object being that you should be fully acquainted with the *causes* of the disease, and that being forewarned you may be forearmed. Never be in a hurry when making a diagnosis, and always attach paramount impor-

* Landon Carter Gray, Lesions from Ear-Disease, Annual of the Universal Medical Sciences, Sajous, 1891.

tance to otorrhœa and ozæna. These you should treat in time to prevent subsequent symptoms of pachymeningitis that might arise, otherwise coma will supervene and you will be utterly powerless to effect any good. To recapitulate: recollect that in otorrhœa, ozæna, and injuries to the skull you are to apprehend pachymeningitis, and that thrombosis of the cerebral sinuses may be one of its results. The patient may die of occlusion of the sinuses, of inflammation of the dura mater itself, or of embolic abscess of the lung. Horsley read a paper before the Neurological Section of the Tenth International Congress on the "Surgery of the Central Nervous System." He advocates "trephining in traumatism of the brain, especially when there is severe and obstinate headache, in *pachymeningitis*,* and in all cases where the existence of a tumor is suspected." He believes that gummata are not amenable to medical treatment, and should be removed by operation, but certainly some experience antagonizes this view. He would tie the common carotid artery in cases of cerebral hemorrhage, if called early.†

The prognosis, of course, is necessarily very unfavorable.

About the treatment there is very little to be said. You may treat the brain-symptoms in this disease as in other forms of meningitis. "If there is reason to suspect the formation of pus between the bone and the dura mater, this may be let out by trephining." (Gowers.)

INTERNAL PACHYMEINGITIS, HÆMATOMA OF THE DURA MATER, OR MENINGEAL BLOOD-TUMOR.

The collections of blood frequently found on the inner surface of the dura mater after death are not due to the rupture of blood-vessels, but are the results of chronic inflammation, as was clearly shown by Virchow.

The condition is usually *bilateral*. Hæmatoma is generally situated near the sagittal suture, is encapsulated, and is nothing but a collection of hemorrhagic exudations.

A membranous layer of tissue exists between the dura mater and the arachnoid which may be adherent to both.

Six or seven layers of this tissue, forming various sacs containing

* Italics my own.

† Packard, Annual of the Universal Medical Sciences, Sajous, 1891.

blood, are found. The blood escapes from the numerous vessels formed in the false membrane of the dura mater, and, as has been hinted before, is effused between the layers of the adventitious membrane.

The encapsulated sacs of blood may be "four or five inches long, two or three broad, and half an inch thick." (Niemeyer.)

Hæmatoma of the dura mater, Gowers asserts, "had previously been ascribed to *primary** hemorrhage, and this view, advocated by Prescott Hewitt in 1845, has been recently revived by Huguenin: the question is still undecided."

The contents of the sac may be filled with fluid or coagulated blood. The brain is more apt to be flattened when the hæmatoma is on one side. Aitken describes hæmatoma of the dura mater as "sanguineous flattened masses, composed of fine layers of fibrin, spread to a greater or less extent over the dura mater, accompanied by small extravasations, which are converted into pigment. By repetition of the process numerous layers come to be deposited one upon the other. Numerous and large blood-vessels form in these layers, and from these vessels renewals of the hemorrhage occur. The disease is chronic, and terminates, after continued cephalic suffering, generally suddenly, with symptoms of apoplexy." The tissue of the new membrane is red at first, afterwards paler. In some places the membranes adhere and form loculated spaces. Virchow affirms that hemorrhage occurs from inflammatory changes in the new membrane.

The disease is exceedingly uncommon. Gowers remarks that "its rarity, at any rate outside asylums, may be judged from the fact that, during the forty years in which the Pathological Society has received the curiosities of metropolitan necroscopy, not a single specimen has been brought before the society from any London hospital."

Niemeyer says that "the disease occurs chiefly in old age, and remarkably often in persons with mental diseases and in drunkards. It appears to develop sometimes as an independent, sometimes as a secondary disease, due to injuries of the brow. In the latter case it is said that years may intervene between the injury and the first symptoms of hæmatoma." (Griesinger.)

* Italics my own.

In other cases, the following factors, to which Griesinger has called attention, enable us, with more or less assurance, to make a diagnosis of hæmatoma of the dura mater: "If circumscribed headaches, gradually increasing to great severity, in the vicinity of the vertex and forehead, be the first and, for a long time, the only trouble of which the patients complain, and if between the appearance of these pains and that of other severe brain-symptoms there be an interval not so short as in acute diseases of the brain and its membranes, but shorter than in most chronic diseases of these parts, particularly in the different cerebral tumors, the first suspicion falls on inflammation of the meninges, particularly of the dura mater, since inflammation of the other membranes has so great a tendency to spread that it is accompanied by *diffuse*, not by circumscribed, headache. We are the more justified in this, as the form of pachymeningitis in question occurs just at the point where the patients complain of pain. If the patient has been mentally diseased before the commencement of the headache, or given to drinking excessively, or if he had an injury of the head, particularly of the forehead, some time previously, there is still more reason for supposing the case one of pachymeningitis, as is evident from the etiology. But we also know that this form of meningitis usually leads to a large effusion of blood, encroaching on the cerebral cavity, and that then the effusion is capsulated on one or both sides of the sagittal suture. Hence, if the headaches be subsequently accompanied by the signs of compression of the capillaries of the cerebrum, by mental disturbances, loss of memory, diminished power of thought, increased inclination to sleep, which finally increases to coma, a slowly developing and usually not pure hemiplegia, after excluding various brain-diseases, we must think of hæmatoma of the dura mater as being in the first rank of those that may possibly be present. Since in hæmatoma of the dura mater there may be reabsorption of the blood and consequent freedom of the brain from the pressure on it, a favorable course of the disease and recovery of the patient speak for hæmatoma in doubtful cases. If the effusion of blood does not take place gradually, as in the course of the disease above described, but occurs suddenly, if it is large and limited to one side, the symptoms are those of an abundant hemorrhage in one side of the cerebrum. On superficial examination it may appear remarkable that, even in

large hæmatomata of one side, there is occasionally no hemiplegia, or else it is very incomplete; but we must bear in mind that hæmatoma occurs just at the place where the increased pressure on one hemisphere is most readily transferred to the other, through the free communication between the two sides in the interior portion of the skull, particularly when the hemorrhage comes on slowly." Among the symptoms of hæmatoma, Griesinger also lays stress on the almost constant contraction of the pupil, and is inclined to regard this as a "symptom of irritation of the surface." In the previous chapter I attempted to give another explanation of the contraction of the pupil (which was also hypothetical) in diseases encroaching on the space above the tentorium. (Niemeyer.)

Aitken considers the possible antecedent existence of syphilis a factor not to be ignored in cases of hæmatoma of the dura mater. It should be remembered that after the initiatory symptoms of irritation have lasted for some time (and after the usual interval following them) symptoms of depression may ensue, with violent and localized cephalalgia as an almost pathognomonic manifestation. The psychical functions are depressed, the memory and the general intellect impaired,—undoubtedly results of the pressure upon the convolutions of the brain, attended by consecutive anæmia or softening, occasioned by the hæmatoma. Somnolence, with a gradually increasing tendency to coma, becomes more and more developed. Occasional attacks of transitory unconsciousness are produced towards the termination of the affection, with the development of a partial hemiplegia.

The principles of *treatment* in hæmatoma of the dura mater are the same as those applicable in cases of cerebral hemorrhage.

RANNEY'S TABLE OF DIFFERENTIAL DIAGNOSIS BETWEEN
EXTERNAL PACHYMENINGITIS AND INTERNAL PACHY-
MENINGITIS, OR HÆMATOMA OF THE DURA MATER.

| EXTERNAL PACHYMENINGITIS. | INTERNAL PACHYMENINGITIS. |
|--|---|
| CAUSES. | |
| Traumatism of the calvaria. | Chronic alcoholism and syphilis. |
| Diseases of the cranial bones. | Acute febrile disorders (fevers, rheumatism, and puerperal diseases). |
| Caries and necrosis of the cervical vertebrae. | Chronic diseases of the heart, tuberculosis, and the paralysis of the insane may be associated with it. |
| Suppurative diseases of the vertebral ligaments. | |

DIFFERENTIAL DIAGNOSIS BETWEEN EXTERNAL PACHY-
MENINGITIS AND INTERNAL PACHYMENINGITIS.—
(Continued.)

EXTERNAL PACHYMENINGITIS.

INTERNAL PACHYMENINGITIS.

CAUSES.—(Continued.)

| | |
|---|--|
| It rarely follows syphilitic or rheumatic conditions of the cranium or erysipelas of the scalp. | Old age. Males more frequently affected than females. |
|---|--|

HEADACHE.

| | |
|--|--|
| Intense and circumscribed headache usually exists. | Periodical headache is commonly produced, gradually reaching extreme intensity whenever the acute form exists. |
|--|--|

CONVULSIONS.

| | |
|---|-----------------------|
| Slight convulsions are common at the onset. | Convulsions are rare. |
|---|-----------------------|

BRAIN-SYMPTOMS.

| | |
|--|--|
| Vertigo, nausea, and vomiting are frequently met with at the onset of the disease. | Weakness of the memory, apathy, somnolence, and delirium are the more common symptoms. |
|--|--|

PUPILS.

| | |
|---|---|
| The pupils are apt to become unequal, if the pressure upon the brain is severe. | The pupils are not necessarily affected, because the pressure of the sanguineous cyst is more limited than that of a pus-exudation. They may be contracted. |
|---|---|

PULSE.

| | |
|--|---|
| The pulse is at first accelerated, but becomes slow and irregular when cerebral compression is produced. | The pulse fails to exhibit the effects of general cerebral compression, except in severe and fatal cases. |
|--|---|

LATE SYMPTOMS.

| | |
|---|---|
| Coma and paralysis follow if cerebral compression or abscess is produced. | Feebleness of the limbs, unsteady gait, and changes in the nutrition indicate the latent progress of inflammation of the brain. |
|---|---|

SYMPTOMS IN COMMON.

Both forms may be associated with headache, convulsions, coma, paralysis syphilitic history.

LECTURE IX.

NEO-MEMBRANES OF THE DURA MATER.

(*Charcot.*)

GENTLEMEN,—In connection with *pachymeningitis*, a study of neo-membranes of the dura mater, recently developed by Charcot, is of great importance, especially as new contributions of this author upon the subject have just been issued from the Parisian press (*Œuvres Complètes de J. M. Charcot*, tome ix., 1890).

Charcot states that it is the generally accepted opinion in France that intra-arachnoidean meningeal hemorrhage results from a rupture of the vessels of the visceral layer of the arachnoid, and that the extravasated blood after a certain time becomes encapsulated by a false membrane which is eventually organized. This new membrane is formed either by a modification of the superficial layers of the arachnoid or by plastic exudation from the parietal arachnoid.

Three hypotheses may be invoked to explain the simultaneous presence of hemorrhage and of membranous neoplasms in the arachnoidean cavity: (1) a hemorrhage occurs and the membranous products ensue; (2) the new membranes are formed before the hemorrhage; (3) the hemorrhage and the membranous products date from the same period.

Charcot asserts that neo-membranes are developed under the influence of spontaneous and inflammatory irritation of the dura mater. On the other hand, far from believing that meningeal hemorrhage antedates the formation of the neo-membranes, he is satisfied that the blood proceeds from the neo-membranes themselves.

In further studying the subject, our author reaches the following conclusions: (1) Neo-membranes may form upon the internal layer of the dura mater, without being necessarily preceded or accompanied by extravasations of blood, while it is especially

noteworthy that the hemorrhage never precedes the formation of the membrane. (2) Meningeal hemorrhage, from many observations, must be regarded most frequently as an epiphenomenon of the development of the neo-membranes; or, more explicitly, the hemorrhage is occasioned by the accidental rupture of vessels previously formed in the new membranes.

The anatomical and pathological history of neo-membranes has been satisfactorily studied only of late. Heschl was one of the first to elucidate the subject. He claimed that they were developed from the connective tissue of the parietal layer of the arachnoid; in this tissue vessels form early, and many layers of connective tissue become thus successively superimposed one upon the other.

In 1856, Virchow described these neo-membranes of the internal layer of the dura mater still more accurately. The dura mater, a membrane apparently little liable to morbid change, is nevertheless susceptible of inflammation. Virchow termed this inflammation pachymeningitis. Pachymeningitis may be internal or external, according as the inner or the outer layer of the dura is primarily or especially involved. Internal pachymeningitis is the form now under consideration; this is generally chronic and exudative. At an early stage a very thin layer of a somewhat fibrinous exudation is poured out, often difficult to detect, but usually recognizable by scraping the surface. By degrees this layer increases in thickness and becomes more and more organized. Connective tissue, nuclei, fusiform bodies and vessels appear. Still later *new exudations* form and are developed in turn into similar tissues. Sometimes from five to twenty superimposed layers may become distinguishable; these form the layers of more recent origin which lie in the closest relationship to the dura mater, and whose organization is consequently most advanced. Usually the membrane which lies in immediate contiguity with the dura mater is but slightly adherent to it. The dura mater on a level with the membranous formation is but rarely altered, with the exception of occasional injection.

In France, M. Brunet (1859) arrived at conclusions similar to those which Virchow had reached three years previously. Brunet pointed out that, independently of other conditions, neo-membranes were found occasionally, post mortem, in cases of gen-

eral paralysis. He thinks that these membranes are developed from the *parietal* fold of the arachnoid, a fold whose existence he admits after discussion of the contradictory opinions of authors upon the subject. Charcot believes, with Kölliker, that this layer has no real existence, at least in the majority of cases; it is only represented by a layer of epithelium. Brunet affirms that neo-membranes are developed from an organizable blastema, concealed by this questionable parietal fold; but, as the elements of the neo-membrane are very probably due to a proliferation of the cells of the dura mater, such an opinion cannot be unhesitatingly accepted.

Calmeil regards the membranes of the arachnoidean cavity as inflammatory products; he observed them in cases of diffused peri-encephalitis.

Virchow's ideas were further elaborated in Germany by Schu-berg, who insisted, among other points, upon the symptomatic-logical phenomena associated with the development of these neo-membranes. Still later, Hasse gave a very complete description of pachymeningitis, in great part based upon his own researches. The pachymeningitic products, according to this investigator, are most frequently found on both sides of the falx of the dura mater; the membrane formed by them is at first very thin, and may become in the end firmly adherent to the dura mater, sometimes covered by new epithelium. Charcot, in a case studied by himself, observed epithelial cells in varying stages of fatty degeneration upon the lamellæ of the neo-membrane. Charcot observes that, if Hasse's observations are correct, an intimate adhesion will be sometimes established between the neo-membrane and the dura mater, and, if at the same time a new epithelium is produced, it may become very difficult to affirm that the fold which covers the dura mater is of pathological origin. In such a case the existence of a parietal fold of the arachnoid may be assumed, although normally this fold does not exist. It is possible that Heschl, Calmeil, Brunet, and other authors who maintain such an opinion may have observed facts of this character, by which they were led to admit the very contestable existence of this lamina of the arachnoid.

Hasse has autoptically encountered pachymeningitis, commencing in pleuro-pneumonia, pleuritis, pericarditis, and acute articular rheumatism, and also, rarely, in variola, scarlatina, and

typhus. He states that, although the pachymeningitic products often undergo a progressive evolution, more or less surely resulting fatally, these untoward sequelæ of inflammation under other circumstances may disappear under the influence of the reactive forces; the membranes will then become thinned, and in such cases, as stated above, may become very intimately adherent to the dura mater and be invested anew by an epithelial layer.

Charcot finally refers to Guido-Weber, who has very recently and exhaustively written upon this subject, like the authorities already cited, from an anatomico-pathological stand-point.

As to the true method by which these membranous products are developed upon the internal surface of the dura mater, all authors quoted concur in teaching that the neoplasms once developed have a great tendency to become the seat of fluxionary movements, a fact explicable as much by the phlegmasial state of neighboring parts as by the rich vascularization of the neo-membranes themselves. Sometimes, from unknown causes, fluxionary movements become unusually pronounced, and the neo-membrane, becoming itself inflamed, may be in its turn the point of origin of exudations and inflammatory products. So it is that in certain cases a more or less abundant serous exudation may occur between the lamellæ of a neo-membrane, even resulting in true serous cysts. Virchow called this form of the disease "pachymeningeal external hydrocephalus." These serous exudations have been often observed by Bayle and Calmeil. Hasse and others have studied them. In subjects of advanced age, Hasse has somewhat frequently encountered serous cysts formed in the neo-membranes compressing the brain and causing a certain degree of atrophy of the cerebral substance. Calmeil, moreover, has observed serous exudations not only between the folds of the neo-membrane, but also between it and the visceral arachnoid. Fibrinous exudations from the neo-membranes also have been observed in various localities.

Sometimes the inflammatory irritation of the neo-membranes becomes more active, and pus is formed: Calmeil and Guido-Weber have reported cases of this sort.

By the arguments submitted, Charcot hopes that a sufficient foundation has been laid to sustain his first proposition,—viz., that neo-membranes of the dura mater are very often developed

as a result of inflammatory processes, without previous meningeal hemorrhage. He goes on to show that in a great number of cases, if not indeed in all, intra-meningeal hemorrhage originates from a neo-membrane of great vascularity and relatively very voluminous, and that, in general, the vessels of these neo-membranes are very thin and predisposed to easy rupture under the blood-pressure.

Neo-membranes once developed tend to increase by superimposition of new layers, from which peculiarity accidents more or less grave may result. Hemorrhage is not an integral or necessary phenomenon of their evolution, even in cases which are otherwise characterized by intensity and gravity. Often, indeed most frequently, the vascular ruptures take place at a definite period, and an extravasation of blood occurs into the neo-membranes. Many things may happen under these circumstances. Sometimes, when the extravasation is very slight, the blood forms small interlamellar ecchymotic collections; at other times, when effused in greater abundance, it separates the layers of the neo-membrane more or less conspicuously, in such manner as to produce real sanguineous cysts, remarkable occasionally for their considerable volume; the walls, finally, limiting the effusion, may burst, and so permit the entrance of blood into the arachnoidean cavity. These various modes of hemorrhage are, in point of fact, phases or differing degrees of a morbid process always in reality the same. The ecchymotic patches result from the rupture of very small vessels, while the sanguineous cysts and intra-arachnoidean hemorrhages, properly speaking, are due to rupture of more numerous and voluminous vessels. In all cases the hemorrhage will be found to originate in the vessels of the neo-membrane.

Such, Charcot believes, is the usual origin of meningeal hemorrhages. It will thus be seen that these hemorrhages, in the greater number of cases, are only an accident of pachymeningitis; a complication which may exist without a definite symptomatic expression, as when the extravasation is slight and disseminated, but which, on the contrary, will reveal itself by a more or less sudden perturbation and by grave phenomena when there is a rapid effusion of a notable quantity of blood.

While the studies of Baillarger were important in subverting the doctrine which located the seat of the effusion between the

parietal layer of the arachnoid and the dura mater, by demonstrating that its real seat is within the layer itself or the epithelial layer which represents it, the well-known hypothesis put forth by this eminent observer concerning the mode of development of these neo-membranes can no longer, in Charcot's opinion, be entirely sustained, in view of the numerous facts in formal contradiction to it.

Before the publication of Baillarger's memoir, Calmeil had already been led to believe that the formation of false membranes might precede intra-arachnoidean hemorrhage; later, in 1835, he enunciated more peremptorily a similar opinion. Although Bayle had not appreciated the fact that meningeal hemorrhage is more often only epiphenomenal, he recognized at least that it is rare to encounter it without the coexistence of a false membrane. It was Cruveilhier, however, who, previously to the most recent observations, distinctly formulated the true theory of intra-arachnoidean hemorrhage. Says Charcot, in the following passage, where he speaks of the formation of certain tumors of the internal surface of the dura mater by the accumulation of caseous puriform matter originating in the processes involving the formation of the pseudo-membrane, "I recognized, Cruveilhier states, that these tumors, ordinarily oblong, had been formed on the internal side of the parietal arachnoid and had resulted from a pseudo-membranous secretion which became organized and formed a variety of cyst, in whose centre was found a material of variable aspect and consistency. *The bloody cysts of the parietal arachnoid are formed by the same mechanism.*" Charcot observes that these few words, hardly more than rudimentary, incontestably express the true theory, obscured, however, and almost lost in the midst of facts which it did not explain, and on this account passed by without recognition. Still later Cruveilhier elaborated and, we may say, definitely formulated this theory.

The following extract is of great importance. "My position as physician for many years at the Salpêtrière," continues Cruveilhier, "permitted me to demonstrate that arachnoidean hemorrhages had as a point of departure a *hemorrhagic pseudo-membranous phlegmasia of the parietal arachnoid*, by the following mechanism: from some cause difficult to determine, a pseudo-membrane adherent to the internal face of the dura mater, and,

consequently, to the parietal fold of the arachnoid, is formed ; a false membrane is never found upon the corresponding arachnoidean visceral layer. This false membrane is sometimes stained with blood, and sometimes contains small hemorrhagic foci in the thickness of its layers. Sometimes it tears and pours into the arachnoidean cavity a more or less considerable quantity of blood. It is this false membrane which is the source of the hemorrhage, and which retains and encysts the extravasations of blood, whose encystment is dependent upon the development of the false membrane, which organizes without contracting adhesions with the visceral layer of the arachnoid, while the blood undergoes all the alterations commonly observed in closed cavities."

Thus it is seen, continues Charcot, according to Cruveilhier's theory, that meningeal hemorrhages, and sanguineous cysts of the parietal arachnoid likewise, originate in a previously-formed false membrane. This fundamental conclusion is also reached by Heschl and Virchow as the result of their interesting researches (Charcot states, in a foot-note, that Virchow in 1856 compared the intra-arachnoidean sanguineous and membranous exudates to the bloody tumors of the ear (*othæmatomata*), and designated them as *hæmatomata* of the dura mater), and by other German authors already referred to in these observations. A perfect correspondence thus exists between the results attained by the French and other foreign authors, as far as the most essential points are concerned, and it is especially this which Charcot so ably contributes to establish. As a matter of priority, Charcot states that Heschl's work deserves recognition: histological study of the subject was inaugurated by him in 1855, several months prior to Cruveilhier's publication.

According to Charcot, Calmeil and Brunet finally admitted that the production of meningeal hemorrhage is dependent in the majority of cases upon the prior existence of neo-membranes, their conclusions being similar to those of Cruveilhier, Heschl, and Virchow. Their conclusions were based upon a great number of original microscopical and clinical investigations supported by pathological histology.

While criticising Brunet, Charcot maintains his work to have been remarkably original in these two latter particulars, although containing some opinions which admit of dispute ; as, for instance,

that the blood in intra-arachnoidean hemorrhage is furnished not by the vessels of the neo-membrane, but by those of the parietal fold of the arachnoid. Charcot states that he cannot admit the validity of this affirmation ; even if it be claimed that hemorrhage from the vessels of the hypothetical layer of the arachnoid precedes that from the dura mater, such an assumption would explain only a minority of the facts connected with the process. The vessels of the neo-membrane exhibit qualities much more in accordance with the supposition which assigns to them the source of the extravasations of blood.

An examination, moreover, of the seat of the effusions would lead us to the same conclusion. If the extravasations be not very abundant, they will be generally found in the space between the folds of the neo-membrane ; if, on the contrary, they are very considerable, they will be more frequently found in the cavity of the arachnoid, or they will be separated from the dura mater by a more or less thick fold of the neo-membrane. Why should we suppose in this case that the blood is furnished by the vessels of the inner surface of the dura mater, thus involving ourselves in unnecessary difficulties ? Were this hypothesis correct, we must first admit that a rupture of the vessels of the dura mater had taken place, followed by a laceration of the internal lamina of this membrane, which, on the contrary, is nevertheless found perfectly intact in the greater number of autopsies. Moreover, if this were so, Charcot claims, it would be necessary to admit, in cases where the effusion is completely encysted, that a rupture of the most external layer of the neo-membrane, capable of permitting the escape of a relatively considerable quantity of blood, could eventually become effaced so as to leave no traces of its occurrence. Finally, where the effusion is found in the arachnoid cavity, it is not merely a thin layer of the neo-membrane, but the entire neo-membrane itself, which must have been detached, distended, and finally ruptured at some point,—a proposition difficult to entertain, as it is in opposition to the facts.

Let us consider, continues Charcot, from another point of view, the structure both of the neo-membranes and of the vessels which permeate them, and we will recognize conditions eminently favorable to the production of hemorrhages of the kind we are studying. These vessels are generally very numerous, and are relatively of

large size, even when capillary ; some of them have a diameter of two- or three-hundredths of a millimetre, yet nevertheless are of the rudimentary structure which characterizes the normal condition of the smaller arterioles or venules ; their walls, in fact, are very thin and show no muscular elements. Whatever may be the degree of organization attained by the tissue of the neo-membrane, and whatever consequently may be its ultimate density and resistance, such a structure, we may say, never acquires the tenacity of the adjacent normal membranes. Hence, concludes Charcot, it is readily conceivable that the vessels as well as the intermediate tissue may easily yield and tear under the effects of blood-pressure ; all the more easily because, in certain cases, fatty deposits form, either in the sides of the vessels or in the thickness of the elements which constitute the neo-membrane, in such a way as to weaken the resistance of all the parts concerned.

When we admit that hemorrhages consequent upon pachymeningitis originate in the vascular apparatus of the neo-membranes, it becomes easy to understand various conditions qualifying the effusion. If the extravasation of blood is considerable, or if the membrane of new formation at a somewhat more advanced stage of its organization is composed, for example, of a small number of fragile and thin layers, the hemorrhagic effusion will dilacerate it, easily breaking through it in many places and finally penetrating into the serous cavity ; this constitutes real intra-arachnoidean hemorrhage. If, on the contrary, the neo-membrane is already highly organized, composed of thick and numerous layers, the blood will force itself between these layers with a facility inversely proportional to the firmness of the adhesion of these layers to each other. In a footnote Charcot adds that the neo-membranes show a very marked tendency to assume a capsular form, and that under such conditions we will almost certainly find within the pseudo-capsule either blood, serous fluid, or a purulent liquid. (Calmeil.) The laminæ of the membranes, more or less strongly adherent to each other, do not separate equally under the pressure of the effused blood, and interlamellar centres are thus produced, which, when somewhat considerable, constitute veritable blood-cysts. Finally, in the cases just considered, where a serous cyst is developed in the thickness of the neo-membrane, it is plain that the vessels of the walls of the cyst itself may rupture, and the extravasated blood become

mixed, in variable proportions, with the encysted serosity. It is quite reasonable to assume that the different varieties of sero-sanguinolent cysts described by authors are formed according to this mechanism.

This theory, which assigns the vascular neo-membrane of inflammatory origin as the point of departure of intra-arachnoidcan hemorrhage, is of extended pathological significance, and serves to explain and define certain non-traumatic hemorrhages which occur in serous cavities widely remote from each other and different in function. Charcot here quotes Cruveilhier once more, as follows: "A great number of facts have demonstrated to me that all the serous membranes are subject to a mode of inflammation that may be termed '*a hemorrhagic pseudo-membranous phlegmasia*.'" Charcot goes on to say that a considerable number of cases of pleurisy and pericarditis styled "hemorrhagic," evidently of this type, can be cited, where the blood has been doubtless furnished by the vascular apparatus of neo-membranes. Certain vaginal hæmatocœles studied by Gosselin may be considered of the same character, because in these cases the blood which is effused in the middle of the serous cavity may emanate from the rupture of numerous vessels with attenuated coats which are distributed throughout the thickness of the membrane of new formation developed upon the vaginal tunic. It is known likewise that some facts pertaining to peri-uterine hæmatocœles have been presented by Tardieu, showing these forms of hæmatocœle to have been dependent upon a circumscribed hemorrhagic peritonitis. A circumstance which also tends to prove that hemorrhagic pachymeningitis and various hemorrhagic neo-membranous phlegmasiæ are allied morbid conditions, is that they may coexist in the same patient. Charcot here quotes from M. G. Weber the case of a man of fifty-one years of age where examination revealed a very manifest pachymeningitis, but where, besides, in the right pleura there was found a considerable hemorrhagic exudation, in the midst of which lay a voluminous blood-clot of recent formation.

To the considerations thus far presented in favor of the views sustained by Charcot may be added those furnished by clinical experience and verified by special sequences and concatenations of symptoms which correspond closely with definite pathological movements. From a symptomatological stand-point, two prin-

cial forms of intra-arachnoidean apoplexy must be distinguished. Sometimes the disease is suddenly developed and comports itself like an accidental affection which could not have been anticipated. Cases of this nature, relatively few in number, seem to be independent of pachymeningitis, and do not directly engage us in their study. On the other hand, it more frequently happens that the apoplectic attack is preceded by more or less pronounced and specially marked morbid phenomena, which can almost always be readily recognized, provided the period of observation be not restricted to the last days of life. The symptoms, according to Schuberg, may be grouped in the following order: in the first period, which often lasts several months, there exist, besides other symptoms, a gradual weakening of the memory and of the intellect, vertigo, and continued or remittent cephalalgia, which may be general or partial; at a later period, in proportion as the intellectual disturbance becomes aggravated, somnolence and apathy appear; speech sometimes becomes slow and embarrassed; the limbs, especially the lower ones, become weakened and the performance of their movements is attended with a sense of uncertainty; partial and incomplete paralyses, more frequently of hemiplegic nature, follow, which exhibit the particular characteristic that they are often augmented and diminished with great celerity; finally, as a terminal manifestation, an apoplectic attack occurs, whose symptoms, moreover, have been perhaps too exclusively and prominently thrust forward in classic descriptions, and which generally causes death in a short time.

Such, Charcot adds, is the mode of evolution of the morbid phenomena in the cases now under consideration. If, as he believes, the exudative inflammation of the dura mater necessarily precedes the hemorrhages, the relation between the symptoms and the lesions will not be difficult to establish; the weakening of the intellect, the cephalalgia, the prostration, the weakness of the extremities, and, in fact, all the symptoms of the first periods, being due to the pachymeningitis. Recrudescences of the sub-inflammatory process of which the dura mater and the pseudo-membranes are the seat, followed by a congestion of neighboring encephalic parts, and perhaps also by reflex influences affecting the system of the cerebral circulation more or less extensively, are finally the organic causes of the attacks of loss of conscious-

ness observed in a certain number of cases. Paralysis occurring at this period, it should be understood, may be transient, and may cease to reappear or not, as it is dependent upon a congestion in its turn subject to augmentation and diminution, either partial or total. Pachymeningitis, moreover, may remain unrecognized when very mild, and also when appearing in the midst of conditions in which the cerebral functions are already disturbed: it is for this reason that pachymeningitis is so frequently ignored during the course of a general paralysis with which it coexists. The apoplectic symptoms of the last stage depend upon a more or less sudden irruption of a variable quantity of blood between the layers of the neo-membrane or into the arachnoidean cavity itself. It is unnecessary to enter further into a consideration of this subject than to say that the symptoms will be found to vary in accordance with the abundance of the extravasation.

From the various points of view occupied in this study, Charcot concludes that the history of intra-arachnoidean hemorrhage must lead to certain therapeutic deductions based mostly upon the following considerations. (1) Intra-arachnoidean hemorrhage, the most common of meningeal hemorrhages, takes place, in the majority of cases, in individuals suffering during a longer or shorter period from pachymeningitis: it then proceeds from a rupture of the vessels contained in the pachymeningeal neo-membranes. (2) Pachymeningitis generally declares itself by a collection of symptoms which, in a certain number of cases at least, is capable of attracting attention to the existence of the affection. (3) The evolution of neo-membranes developed under the influence of pachymeningitis sometimes terminates by a retrograde process, at the end of which they may disappear. It follows that, if significant indications are observed adequate to excite a suspicion of a neo-membranous inflammation of the dura mater, the further development of the inflammatory process should be prevented by all the methods ordinarily adopted to check chronic inflammation, and such a course should be all the more insisted upon because this affection, as has been said, even after the formation of organized products, may retrograde and sometimes completely disappear. In other words, by instituting the radical treatment of pachymeningitis we may forestall and prevent the occurrence of intra-arachnoidean meningeal hemorrhage.

LECTURE X.

GENERAL MENINGEAL HEMORRHAGE.

MENINGEAL hemorrhage is rare except as a result of traumatism. Effusions of blood in the subarachnoid space, or between the dura and arachnoid, are due for the most part, as Niemeyer claims, to dilacerative irruption of cerebral hemorrhages, bursting of aneurisms or of degenerated arteries; in many cases their origin cannot be determined.

According to Gowers, in meningeal hemorrhage blood may be extravasated (1) outside of the dura, separating it from the bone (*extra-dural hemorrhage*); (2) beneath the dura, into what was regarded as the sac of the arachnoid when it was thought that a parietal layer of the arachnoid lined the dura (*sub-dural hemorrhage*); (3) beneath the arachnoid, between it and the pia (*sub-arachnoid hemorrhage*). The blood may come from the arteries, veins, or sinuses of the dura, or from the vessels of the pia.

Gowers enumerates the chief causes of these hemorrhages as follows. (1) Injury that causes fracture of the skull or laceration of the pia. Extensive hemorrhage usually proceeds from the meningeal arteries or sinuses. The blood may be outside of or beneath the dura. (2) Aneurisms of the larger arteries of the base or surface. (3) Rupture of an intra-cerebral hemorrhage. (4) Meningeal hemorrhage occurs, apart from visible aneurisms, under the same conditions (age, chronic kidney disease, etc.) as hemorrhage elsewhere in the brain. It is also met with in some chronic diseases with hemorrhagic tendency, as purpura, leucocythæmia, and the malarial cachexia. (5) It occurs occasionally in the insane, especially in the subjects of general paralysis. (6) During birth it may result from the compression of the skull, especially in cases in which the head is born last. (7) Spontaneous hemorrhage from a meningeal vein has been observed, but is excessively rare.

The autoptical appearances in meningeal hemorrhage, says this author, differ according to its seat and amount. There is a layer of blood upon the arachnoid or in the sub-arachnoid space, sometimes in both localities. The blood accumulates especially in the sulci and depressions of the cerebral surfaces, and is generally most abundant at the base and sometimes limited to that region. If effused in considerable quantity over the convexity, the convolutions may be distinctly flattened.

The resulting symptoms quite often do not become manifest for some hours, as the blood escapes more or less slowly from the ruptured artery. This is very significant from a diagnostic point of view, and you will frequently meet with cases of severe head-injury where but few indications of trouble occur for twenty-four hours, when the development of cephalalgia and gradually increasing coma, accompanied or not by epileptiform convulsions and paralysis, will unerringly point to the nature of the mischief. Such hemorrhages are necessarily *diffuse*, and the resulting symptoms are not regional. All authors agree in the statement that the apoplectic phenomena are most highly pronounced, and that this form is quite fatal. Hemiplegia is usually absent, as the hemorrhage is not restricted by definite anatomical limitations, so that when paralysis occurs we necessarily expect it to be general. In all diseases affecting the convexity of the cerebral convolutions, epileptiform convulsions are common. Such convulsions, observed during profound coma without hemiplegia, are pathognomonic of meningeal hemorrhage.

In some cases, says Niemeyer, the coma is preceded by severe headache and vomiting, and in others by general convulsions. He also asserts that, "since these symptoms, particularly the latter, occur only exceptionally in cerebral hemorrhage, and are often seen in extensive disease at the convexity of the hemisphere, they, in connection with the absence of all signs of hemiplegia, enable us to decide with greater certainty that the case is not one of cerebral but of meningeal hemorrhage."

The symptoms of meningeal hemorrhage vary greatly according to its origin. If the hemorrhage is due to a rupture of a large aneurism at the base of the skull, there will be intense coma; if to that of smaller vessels, the effusion of blood, Gowers asserts, will be preceded by such prodromata as giddiness, headache, and vomiting.

The same author calls particular attention to the fact that when the hemorrhage is of traumatic origin the effect of the injury obscures the initial symptoms, because "in these and similar cases, in which the escape of blood is gradual, the patient may recover consciousness and continue his occupation for some hours, or even for a day or two, complaining only of headache, and then gradually become somnolent and pass into a state of coma."

In meningeal hemorrhage, Gowers considers the convulsions the most prominent symptom: they may be general, unilateral, or local. Rigidity of a limb, he states, is rarer than in meningitis; delirium or mental apathy may exist; the pupils may be contracted, dilated, or unequal. The variation of the symptoms in different cases is very great.

Hammond quotes Prus as "attempting to draw a symptomatological distinction between sub-arachnoidean and intra-arachnoid-ean hemorrhage. . . . But most authors doubt if the discrimination can in reality be made during life. Valleix declares that the difference is of greater anatomical than symptomatological importance, and Durand-Fardel admits that it is difficult to present a characteristic view of the course and phenomena of sub-arachnoid-ean hemorrhage."

Meningeal hemorrhage is much more common in adult life than in young people. Besides traumatism and disease of the vessels of the brain, extreme heat, venereal excesses, excessive mental exertion, alcoholism, and obstructions to the venous outflow from the head, etc., have been enumerated as exciting causes. One of the most effective predisposing causes of meningeal hemorrhage is the peri-arteritis which leads to the development of miliary aneurisms, so graphically described by Charcot and Bouchard. Hæmophilia and gout are also mentioned as causes. The disease is most frequent in the two extremes of life, especially in newborn children as a result of instrumental labor.

The *prognosis* in nearly all cases is very grave: recoveries are of the rarest occurrence.

I must not forget to remind you of Erichsen's form of progressive extra-dural hemorrhage, a variety which is often misconstrued or even escapes detection. By a blow from a blunt instrument, as a sand-bag, or by a fall upon the head, the dura may be detached from the inner surface of the cranial vault, and

hemorrhage may occur which does not necessarily proceed from the rupture of the meningeal artery or its main branches. The bleeding has its source in the laceration of the nutritive vessels of small size which pass from the dura to the bone. As blood is poured out, the dura is still further detached and the collection of blood increases in size. Here the symptoms of compression gradually supervene, and after a period of many hours the patient becomes soporose and comatose. Recollect that all this commonly occurs without fracture or even external contusion.

The *treatment* is similar to that of cerebral hemorrhage, which will be particularly considered in the second volume of this work. I fully agree with Hammond that, "in those cases where the symptoms show that the clot is confined to a small area, trephining and the subsequent removal of the clot may be successfully accomplished."

LECTURE XI.

CHRONIC CEREBRAL MENINGITIS.

Hammond's Classification—Brown-Séquard's Views, etc.

GENTLEMEN,—After careful thought, I have concluded to adopt Hammond's classification, and to speak of this subject under two captions,—viz., *Chronic Vertical Meningitis* and *Chronic Basilar Meningitis*.

For much of the spirit of this lecture, and for many quotations contained in it, I am indebted to a "Treatise on the Diseases of the Nervous System," by Dr. Hammond, ninth edition, 1891.

The author just named, referring to the division of the subject into *Chronic Vertical Meningitis* and *Chronic Basilar Meningitis*, states that "the terms, being applied respectively to chronic inflammation of the membranes of the superior surface or vertex of the brain, and chronic inflammation of the membranes of the inferior surface or base of the brain," constitute, according to his ideas, the proper study and conception of the comprehensive subject of chronic cerebral meningitis.

CHRONIC VERTICAL MENINGITIS.

An acute attack of meningitis may have preceded the chronic form.

ETIOLOGY.

The causes of chronic cerebral vertical meningitis are but too frequently obscure. Blows upon the head, diseases of the bones, syphilis, rheumatism, exposure to extreme heat, emotional and mental influences, are all powerful factors in its production. While alcoholism is perhaps the most general and influential cause of this disease, syphilis is perhaps its next most common one, but I agree with Hammond that the latter malady "acts preferably

upon the basilar portion of the membrane." Gout in some instances has been known to produce it.

ANATOMICAL APPEARANCES.

The membranes are greatly injected and sometimes adherent to each other; there is increased proliferation of connective tissue; the meningeal coats are thickened and less transparent than normal.

Fibrinous exudations are frequently encountered both on the convexity of the membranes and on the brain itself.

The sub-arachnoidean fluid is increased in quantity and may be discolored by effusion, and pus may be found in the arachnoid cavity and in the meshes of the pia. Gelatiniform exudations and false membranes are not uncommon. Cysts containing blood and serum are also encountered. The ventricles at times are found distended and their ependyma inflamed. The cortex cerebri may be inflamed, softened, or adherent to the pia-arachnoid.

SYMPTOMS.

It is a notable fact that the symptoms of chronic vertical meningitis at times greatly resemble those of the disease known as "progressive general paresis of the insane," a malady to be described in a subsequent lecture.

Headache, a prominent symptom common to all forms of meningeal inflammation, is almost always present. The pain is usually *diffused*, more or less *persistent*, though occasionally remitting. It is commonly felt in the forehead and vertex, and is aggravated by movements, especially by lowering the head, and by intellectual effort. Its principal characteristic is persistency. Of course its intensity is not so great as in the more acute forms of meningitis.

Lethargy and stupor, weakness of the limbs, vertigo, and tremor constitute a well-known symptom-group. The sphincters are more or less affected. Articulation may or may not be impaired. Spasms of special groups of muscles frequently occur. The mental faculties are often dulled. Convulsions sometimes occur. The cranial nerves are not so frequently involved as in other forms of meningitis, especially the basilar form.

Disturbances of sensation, notably neuralgic pains in various parts of the body, are not uncommon.

The weakness of the limbs appears to be a form of paresis rather than of paralysis proper, although absolute hemiplegia has in some cases been noted. Optic neuritis is rare. Hammond, quoting Dr. Allbutt, observes, with respect to the optic nerves in drunkards affected with meningitis of the convex surface of the brain, that they are "often degenerated, and the vessels injected, but these effects do not seem to be due to any meningitic process."

Should the cortical substance become seriously involved,—which does not often happen,—the mental symptoms will become more prominent and their deterioration but too evident.

The general health fails in some cases, but in many instances is little affected.

The constipation and vomiting occur which are associated with nearly all forms of meningitis. In some cases loss of sight is produced by pressure on the optic nerves.

If the case grow worse, all the symptoms will converge towards coma and death.

The duration of the disease varies from several months to several years.

An interesting though infrequent occurrence should be mentioned here. An *intermittence* of the symptoms often exists, and is accompanied by what Dr. Fox has particularly noted (a fact Dr. Hammond has also commented upon in his excellent article on chronic cerebral meningitis),—namely, "the *lightness* of the phenomena when compared with the *severity* and *extent* of the lesions."

These remissions in the symptomatic manifestations are obscure and very difficult to explain.

A case has come under my observation illustrative of these statements. It was one of a physician, presenting many symptoms of chronic meningitis, with frequent severe exacerbations, accompanied by complete forms of paralysis, whose frequent remissions were at times so marked that he was able to attend to his practice when the exacerbations of his affection had disappeared.

Another case, probably of syphilitic origin, for nearly a year presented no symptoms, except a persistent, intense, diffused headache, with some slight impairment of sight; yet I feel sure that extensive and complicated lesions existed.

I beg leave to quote the following important views of Brown-Séquard in this connection :

"If we compare the symptoms produced by an irritation, such, for instance, as that of one of the lungs, or of the part of the base of the brain where the trigeminal nerve is inserted on the pons Varolii, or of the cerebral meninges, with the symptoms caused by an irritation of any part of the brain, we find that in either of the groups of cases we compare there may be paralysis produced in the corresponding or on the opposite side of the body, and, besides that, almost any of the symptoms of disease of the brain. On the other hand, we find also, and in many cases, that no symptom of brain-disease will appear, notwithstanding an organic disease in the cerebral lobes, in the base of the brain, in the cerebral meninges, or in the lungs.

"Led by facts like the above, and by many others, I have necessarily come to the conclusion that there are nervous elements in the various parts of the cerebral lobes, in the corpora striata and optic thalami, in the crura cerebri and other parts of the base of the encephalon, which, like the nervous elements of the cerebral meninges, of the various viscera, the skin, the mucous membranes, the trunks of nerves, etc., have, when irritated, the power of producing on some part of the nervous centres—either at a short or at a very great distance from the seat of the irritation—changes in the normal state of activity of that part.

"These changes may consist (a) in *cessation of activity* ; . . . (b) in the manifestation in a morbid form of the activity of certain parts of the nervous centres. . . .

"From all of the above statements and from the study of every symptom of brain-disease, I have drawn the conclusion that all the parts of the brain resemble the peripheric parts of the nervous system, in being able, under irritation, to act on any other of its parts, modifying their activity, so as to destroy or diminish or to increase and to morbidly alter it." *

In another place the same author observes, "*There is no relation whatever between the extent of a lesion in the brain and the production of symptoms.* . . . This can be expressed in other and

* Brown-Séquard, On the Mechanism of Production of Symptoms of Diseases of the Brain.

perhaps more forcible words: *there is no relation whatever between the extent of a lesion in the brain and the symptoms that may be caused by it.* If the symptoms were due, as is admitted, to the loss of function of the part altered or destroyed in the brain, or to *immediate* effects of the irritation of such a part, there would be a constant relation between them and the disease, so that the intensity and extent of the symptoms would be in proportion with the intensity and the extent of the alteration. . . .

"The mechanism of production of symptoms of brain-disease, as I have already said, and as I will, I hope, succeed in showing in a special paper, is identically the same, whether the cause is in the bowels, or in any thoracic or abdominal viscus, or in the meninges of the brain, or in any part of the brain itself. The symptoms are not the *immediate* or *direct* effects of either the cessation of function or a morbid action of the part diseased. Wherever is the lesion which is the prime mover in the causation of symptoms, it entirely or at least partially produces them *mediately* or *secondarily*, or *indirectly*, and through the agency of an irritation starting from the seat of the lesion and acting on other parts which in a direct way give rise to the morbid manifestations. . . .

"A lesion of the brain can produce a cessation of symptoms of brain-disease. There are facts on record showing that symptoms of brain-disease may disappear after considerable lesions of the brain. Thus, in a case of insanity and epilepsy there was almost a complete cure after a fracture of the cranium and the issue of a notable quantity of brain-tissue. . . .

"*An immense variety of symptoms in different individuals may be caused by a lesion in one and the same part of the brain.* Symptoms of brain-disease vary immensely, not only in intensity, as stated already, but also in their kind, although the lesion producing them occupies the same place. . . . The only rational explanation is, that, in the same way that an irritation of any peripheral nerve may either be insufficient to produce a remote effect, or quite able, on the contrary, to produce the most varied effects, a disease in any part of the brain may also be the starting-point of either an inefficient irritation, or act by means of that irritation on distant parts and produce through them either kind of symptoms."

DIAGNOSIS.

It is often impossible to make an absolute diagnosis in chronic meningitis; especially is the difficulty magnified when we attempt to differentiate between the last-named affection and encephalitis, and more particularly when we consider that these two affections sometimes coexist.

It is only by a careful study of its *etiology*, the sequence of symptoms, the course of the disease, the prominence of *pain* as a symptom, and collateral facts, that we can solve the diagnostic enigma.

The absence of marked mental symptoms also favors a diagnosis of *meningitis*; so does the occurrence of delirium and convulsions.

PROGNOSIS.

The prognosis is always serious. The most favorable cases are undoubtedly those of *specific* origin, in which radical antisyphilitic treatment is often highly beneficial.

TREATMENT.

Mercurials and iodide of potassium form our main reliance in these cases. The bromides and ergot may be given in the earlier stages. The bowels should be regulated by laxatives. Counter-irritants, and especially the chronic pustulation of the shaven scalp with croton oil, may prove of some service. The patient's diet should be regulated. Stimulants should be eschewed. Emotional excitement and mental fatigue should be guarded against.

An excellent method of administering iodide of potassium is in *concentrated* solution, thus:

R Potassii iodidi, $\mathfrak{z}\text{i}$;

Aquæ destil., q. s. ad $\mathfrak{f}\mathfrak{z}\text{i}$.—M.

S.—*Each minim* of the above solution will represent *one grain* of the salt.

(N.B.—From ten to twenty minims would represent an average dose, which may be increased a drop or a minim each dose in cases where a progressive increase in the dose is desirable.)

CHRONIC BASILAR MENINGITIS.

Probably the most common cause of this disease is *syphilis*; *alcoholism* ranks next. Excessive anxiety and emotional disturbances of all kinds are among its alleged causes.

Hammond states that "next in point of frequency come atmospheric vicissitudes, blows on the head, and attacks of other diseases, as scarlet fever, and especially epidemic cerebro-spinal meningitis, and suppurative otitis. Men are more subject to it than women, and adults more than children. Frequently no cause can be assigned."

SYMPTOMS.

Headache, as I have frequently reiterated in the preceding lectures on the forms of meningitis, is an early, frequent, persistent, and very prominent symptom.

Epileptiform attacks very often coexist with the cephalalgia. Hammond insists upon this point, and adds, "There may be convulsive movements of a limb, a group of muscles, or a single muscle, unattended with loss of consciousness. Again, there may be tonic spasms of the muscles of one or more of the extremities, especially of the arms; or the muscles of the neck may be similarly affected, causing the head to be fixed in an abnormal position. The individual muscles of the face are not usually involved."

Paralysis sooner or later occurs. It may be of a hemiplegic character, of the ordinary cerebral type. Facial palsy and difficult articulation are sometimes noted.

Some, or nearly all, of the motor nerves of the eye may be involved.

A common occurrence is *unilateral* paralysis of the *third* nerve, resulting in *ptosis*, *defective accommodation*, *diplopia*, *dilatation of the pupils*, and *external strabismus*. This paralysis is conceded, by all authorities with whom I am acquainted, to be particularly characteristic of meningeal and intracranial syphilis. On this point I cannot lay too much stress, and I shall advert to it again in subsequent lectures. The paralysis may be complete or partial.

Hammond asserts "that in a few cases the only indication of the affection of the third nerve is dilatation of the pupils."

Paralysis of the fourth and sixth nerves, vertigo, mental confusion, and aphasia, are frequently encountered.

It has been my sad experience to witness a corroboration of Hammond's observation, that in a few isolated cases pain of the

most extreme severity and persistency, "almost driving the patient to suicide," may exist for a long time as the *only* symptomatic indication of a latent basilar meningitis. I am at present attending a gentleman in whose case seven prominent physicians were unsuccessfully consulted, before he placed himself under my care, for the above-described condition. I have treated him during a long period without affording him the slightest relief. Galvanism, cinchonism, opiates, the application of the thermo-cautery to the nucha, mercurial preparations, iodide of potassium in half-ounce doses daily, antipyrine, chloral, sulfonal, have all failed to produce any mitigation of the extreme pain. Three or four hours of fitful slumber have hardly been procured, so obstinate has been the insomnia resulting from the intense headache. At no time has he complained of localized cephalalgia, which, on the contrary, has been erratic. Its diffusion has been as remarkable as its persistent duration. There have been no evident remissions of the headache. It has been, perhaps, more particularly prominent in the occipital and frontal regions, sometimes extending to the face. Repeated examination has failed to detect optic neuritis. There is entire absence of all other symptoms. There is no *specific* history of any kind. The case is a real *opprobrium medicorum*, as far as tentative therapeutic results are concerned. It has excited my deep sympathy; I really dread the patient's visits, and yet I doubt not that it is one of those exceptional cases of chronic basilar meningitis so graphically described by Hammond.

Anæsthesia, limited or diffused, accompanied or not by disturbances of the kinesodic zone, may or may not appear.

Disturbances of vision are frequent, often appear early, and may be of a general or a special character.

Disturbances of the cranial nerves are very common. Optic neuritis often coexists. Vision is sometimes entirely lost. Hearing may be impaired or destroyed. The mind is not primarily affected to a notable degree. Mental depression not infrequently manifests itself, and the processes of intellection are at times defective.

Mutability of *symptoms* becomes evident when there exists a transference of *seat* in chronic basilar meningitis. This change of location is expressed by alterations of the symptoms, which then become migratory and are somewhat modified.

ANATOMICAL APPEARANCES.

The inflammation in chronic basilar meningitis is much more limited and circumscribed than in chronic vertical meningitis, and, as Hammond remarks, "it may be restricted to a portion of the membranes not larger than a dime."

The membranes are usually thickened and more or less opaque. More or less serum will be found in some places; in others, a thick, gummy, or puriform collection. The exudations may become organized, and are sometimes adherent. The injection of the pia-arachnoid is in some places intense. The effusion may be sub-arachnoidean, may lodge in the meshes of the pia mater, or may produce ventricular distention.

The pia mater sometimes contains deposits of a whitish-gray fibrinous substance, following the course of some of the cerebral vessels, and Hammond asserts that it is particularly found "over the chiasma of the optic nerves, the tubercula mammillaria, and the anterior perforated spaces."

The optic and olfactory nerves may be atrophied.

When the arachnoid and the pia are adherent, the latter membrane is in some instances so firmly glued to the cortical substance of the brain also that it cannot be separated from it without laceration.

Whether the exudation be fibrinous, serous, or purulent, it is generally situated in the meshes of the pia mater, in the layers of the arachnoid, or in the sub-arachnoid spaces; but in chronic basilar meningitis it is more particularly found at the base of the brain, and, as Hammond observes, "its usual situations are the chiasma of the optic nerves, along the course of these nerves, on the tuber cinereum, the corpora mammillaria, and between the crura cerebri. Sometimes it extends anteriorly along the course of the olfactory nerves, laterally into the fissure of Sylvius, and posteriorly as far as the pons Varolii and medulla oblongata."

There can be no doubt that chronic basilar meningitis is frequently of syphilitic origin, although Gintrac, as quoted by Hammond, "doubts its existence, though he admits its possibility:" the latter authority, however, does not concur in this opinion.

Hammond states that "in the syphilitic form of the disease it is a matter of some doubt whether the gummy exudation is the

result of a specific inflammation of the membranes, or whether the inflammation is excited by the presence of the new formation."

Fox, quoted by Hammond, states it as his opinion "that it is at best an open question whether meningitis ever occurs independently of syphilis, rheumatism, alcoholic poison, tubercle, anæmia, or mechanical irritations."

Gummy tumors are usually found at the *base* of the brain. They may be either circumscribed or diffused; "but ordinarily they are more diffused, and are accompanied with the phenomena of inflammation, a fact which seems to distinguish them from the true tumor," says Hammond, who follows Virchow in regarding this condition as a "gummy inflammation."

The seat of syphilitic basilar inflammation is the same as that of the other varieties of chronic basilar meningitis. "Hence it is," reiterates Hammond, "that the nerves lying at the base of the brain, and especially the *third pair*, are so liable to be implicated. This latter, from its exposed situation, running, as it does, from the *crura cerebri* to the orbit, can scarcely escape being involved in the morbid process." Focal chronic meningitis is usually syphilitic, and is often associated with disease of the arteries. The nerve-sheaths are often reddened. Minute hemorrhages may occur therein, and the fibres may be degenerated. The inner surface of the *dura mater* may be congested, covered with lymph, and adherent to the *pia-arachnoid*. The choroid plexus and *velum interpositum* may be inflamed. The third ventricle, aqueduct, and fourth ventricle are often distended; their communication is often obliterated by accumulations of lymph in the neighborhood of the aqueduct of Sylvius and valve of Vieussens.

PROGNOSIS.

Like all other forms of meningitis, chronic basilar meningitis is very fatal, and its prognosis is most unfavorable. Syphilitic cases, if subjected early to an earnest and radical specific treatment, frequently improve, under circumstances apparently most unpropitious.

Alcoholic cases, if the lesions be not too far advanced, may improve, provided the habit be controlled. Traumatic cases are generally fatal.

When the disease is due to mental and emotional causes, it is

susceptible of amelioration, if such baneful influences be regulated or abrogated.

Recurrence of the attacks, their *duration*, and *age*, especially the two extremes of life, all bear unfavorably upon the prognosis. It is especially asserted, by Hammond and other writers, that when the disease is the result of an extension of aural inflammation, an unfavorable termination is to be anticipated.

DIAGNOSIS.

The existence of cerebral tumors is betrayed by symptomatic phenomena very apt to mislead in the diagnosis of this disease; this point will be especially considered in our lecture on such tumors. We agree with Hammond that "the symptoms of chronic basilar meningitis are less pronounced than those of tumors at the base of the brain, while, at the same time, they are ordinarily developed with greater rapidity. Another mark of difference is the fact that tumors non-syphilitic in character do not yield to remedial measures, while chronic basilar meningitis often does, and is generally mitigated by proper treatment."

"Chronic softening, arising from thrombosis of the basilar arteries and diseases of the capillaries, is sometimes confounded with chronic basilar meningitis." (Hammond.)

Virchow, as quoted by Hammond, "goes so far as to doubt if, even where after death we find only meningitis, the condition has not been preceded by a gummatous affection which has disappeared."

TREATMENT.

Mercury, the iodide and bromide of potassium, and ergot are indicated in the treatment,—the two latter in the earlier stages. Electricity and strychnine may be resorted to in the paralytic complications of subsequent and more advanced stages. In syphilitic cases mercury, especially by inunction, should be systematically administered. Mercury will prove beneficial in proportion to the recency of the syphilitic manifestations. The dose of iodide of potassium in these cases should be *gradually* increased to half an ounce or an ounce, or even more, daily. It should be administered in a goblet of milk, to prevent gastric irritation. In *septic* meningitis, Gowers extols the perchloride of iron.

GENERALITIES.

In *chronic alcoholic meningitis*, which affects chiefly the convexity, the most prominent symptoms are headache, slight optic neuritis, more or less delirium, and mental failure. This disease runs a very chronic course, and its symptoms are complicated with those proper to alcoholism.

In chronic meningitis of the *syphilitic* variety, Gowers says that "the inflammatory tissue has undergone fibrous transformation, and a thick layer of tissue, tendinous or cartilaginous in aspect, extends over a certain region, more often at the base than at the convexity, surrounding and compressing the nerves, and uniting the various membranes. The thickness of the layer may be as much as a third of an inch; the dura mater is often also thickened."

The *focal* form of inflammation in chronic meningitis in adults is nearly always syphilitic.

I fully concur with Gowers that "in such a case it is useless cruelty to suggest to a husband, by questions regarding his past history, that he may be the unconscious cause of his wife's malady. It is unlikely that by such questions syphilis can be absolutely *excluded*, and unless it can be excluded the anti-syphilitic treatment in such a case is the first duty of the practitioner. Moreover, if syphilis can be excluded, the treatment suitable for syphilis remains the most promising for a simple inflammation."

"The pia-arachnoid differs from most other membranes that enclose viscera, in its separation into two layers. Nevertheless, it is commonly regarded as a serous membrane, and it presents some analogies to other serous membranes in its pathological liability, but also wide differences from them. Like the pleura, it is prone to spontaneous inflammation, but the most common cause of primary pleurisy, exposure to cold, seems to have little influence in exciting meningitis. It is the seat of specific processes more frequently than any other serous membrane, and this, together with its liability to suffer in states of blood-poisoning, must be regarded as its chief pathological characteristic. The process of inflammation also presents some peculiarities in the cerebral membrane. The tendency to the formation of lymph is smaller, and of pus is greater, than in the case of the pleura or the pericardium.

Embolic processes may play a part in the generation of some forms of septicæmic inflammation, but it is probable that the circulation of the septic matter in the blood, not necessarily organisms, suffices to excite in the membranes the inflammation to which they are prone. The origin of the miliary tubercles of the meninges is a problem that belongs to general pathology." (Gowers.)

In chronic cerebral meningitis the significance of the headache depends upon its intensity, persistency, and diffusion. To use a paradoxical expression, its diffusion is in direct correlation with its fixity. Delirium, spontaneous vomiting, and convulsions are often prominent and early symptoms, in conjunction with headache.

The symptoms, of course, vary, and but too frequently are protean in their manifestations; there is no symptom of meningitis that may not at times be absent, and it may safely be stated that there is absolutely no pathognomonic symptom of chronic meningitis.

Attention to the etiology of the disease is of paramount diagnostic importance.

In cases where there exists a tubercular heredity, a meningitis will almost certainly be of the prevailing diathetic type.

Gowers says, "the discovery of tubercles of the choroid renders the nature of the inflammation certain."

The same author remarks, *speaking of chronic meningitis in general*, that "if the inflammation is at the convexity, the probability of its tubercular nature is considerable in childhood and youth, but in adult life such inflammation is probably not tubercular. Under twenty years of age there is a presumption, in the absence of other causal indications, that any meningitis is of tubercular origin, but over forty there is a presumption against this, which increases in weight as life advances."

It is well to bear in mind that in cases of acute general diseases in which head-symptoms exist we shall avoid the danger of confounding these with the symptoms of true meningitis if we remember Gowers's quotation from Sir William Jenner, that "when they are the result of pyrexia, the headache ceases when the delirium begins; in meningitis the headache continues and coexists with the delirium."

It is also well to bear in mind the fact that while optic neuritis

sometimes accompanies acute specific diseases, it is more apt to follow in their train than to coexist with them.

Optic neuritis, when complicating meningitis, is usually much less intense than in cases of tumor.

Meningitis has sometimes been confounded with hysteria. This multiform affection may be associated with any organic disease of the nervous system, but must be recognized as a complication of meningitis or reversely, under the guidance of broad principles of diagnosis. This point has been most ably discussed by Dr. Seguin, of New York, in a recent able monograph. Where doubt exists, the development of the symptoms of organic disease must be carefully watched for and minutely scrutinized. Fever and optic neuritis, when present, will be symptoms of material import in making a diagnosis.

Gowers asserts that "strabismus in hysteria is always convergent and attended by spasmodic contraction of the pupils. Divergent strabismus or inequality of pupil is certain evidence of organic disease, and as much so if it is transient as if it is permanent. Retention of urine may be due to hysteria, but incontinence never is."

Gowers cautions us to avoid mistakes in the differential diagnosis between chronic cerebral meningitis and acute double otitis of children, in which we may observe headache, vomiting, fever, delirium, giddiness, convulsions, and deafness. He suggests that in such cases the labyrinth is chiefly affected. Care in diagnosis in such a contingency is all the more necessary because in some cases of acute double otitis optic neuritis may be developed. He adds that "the difficulty is increased by the fact that the internal ear may be inflamed secondarily to meningitis. Such secondary otitis has been observed in cerebro-spinal meningitis, but it is very rare."

The presence or absence of optic neuritis in meningitis and in brain-tumors does not always throw as much light upon the case as we might wish.

"The significance of optic neuritis as a diagnostic and prognostic sign in cases of cerebral tumors is alluded to by Mr. Horsley in his interesting communication, in which he details his experience in cerebral surgery. In two of the three cases on which he operated it appears that there was no optic neuritis, and in these two

recovery ensued. In the third, a case of cerebellar tumor, there was optic neuritis, and this patient died. To these may be added, besides those reported in this paper, Dr. Hughes Bennett's and Mr. Godlee's case, in which optic neuritis was present, and a case of cerebellar tumor with neuritis recently operated on by Mr. Bennett May. Both of these were unsuccessful. Thus, out of five cases, the two which had no optic neuritis recovered, while the three which had optic neuritis died. It may be that this coincidence is accidental, for recoveries take place in some cases of head-injuries and cerebral abscesses in which neuritis has occurred; but still its presence must be taken to indicate some complication which is probably harmful, and makes it important that, if possible, tumors of the brain should be recognized before the onset of optic neuritis, and, therefore, without its diagnostic aid."* The same may be said of meningitis, cerebral abscess, and other intracranial affections, in which evidences of optic neuritis are so frequently and carefully sought for by investigators.

It should always be borne in mind that meningitis may be excited as a result of suppuration in the middle ear, while as yet no discharge whatever has occurred.

* Surgery of the Brain, Senn, Sajous's Annual of the Universal Medical Sciences, 1888.

LECTURE XII.

INSANITY.

Insanity a Disease of the Brain—Its Origin—Location—Predisposition—Insanity Hereditary—Definition—Unconscious Cerebration—Moral Insanity; Examples—Rules for Ascertaining Insanity.

GENTLEMEN,—I propose to-night to enter upon the subject of insanity. We must not leave this disorder unstudied on account of its infrequency, for the young practitioner must expect and be prepared to meet all disorders. It is not a disease of the mind, but a peculiar affection of the brain. You will undoubtedly be called upon to express your views of its nature, and will naturally be expected to possess some knowledge of its leading features. The study of insanity is a matter pertaining to the general practitioner, for this malady is as distinctly a disease of the brain as any other affection of that organ. It consequently demands his attention as cogently as any other disorder of the human body. Though a true disease of the brain, it does not invariably originate in that organ; indeed, it may start almost anywhere else, as in the diseased uterus in the female, or in some morbid condition of the colon, and in either case may disappear if proper treatment be addressed to its source.

When a man is insane there is always a disturbance of the normal working powers of the brain. Insanity may have the greatest multiplicity of primary causes. In the majority of cases, I believe, insanity originates at a greater or less distance from the brain, and hence is mostly a secondary affection, and but rarely idiopathic.

This affirmation constitutes one step towards a proper elucidation of this subject, for it implies that insanity is not necessarily a stigma, as was formerly supposed; nor is it, metaphysically speaking, a disorder of the mind, with accompanying mental disturbances of a mysterious character. It is an affection of the

brain, which, though not always originating in that organ, *invariably has its seat there.*

You will not find it difficult to appreciate these facts if you recollect that the brain is the supreme centre, presiding over all the other parts of the nervous system and of the animal economy. Indeed, we can hardly conceive of any constituent atom of the body, no matter where found, which is not in more or less intimate relation with the brain or the cerebro-spinal system of nerves. It presides over multitudinous atoms, its influence is felt throughout the body, and there is not a muscle, nerve, artery, or vein, nor even the smallest histological element in the human system, which is not permeated by the peculiar nervous force or influence emanating from the "supreme cerebral ganglia." How easy, then, is it to understand that the functions of the brain may be more or less impaired by the presence of disease there or elsewhere, especially where any predisposition to insanity exists!

In all disorders leading to insanity this predisposition undoubtedly exercises a great influence and plays a most important rôle. That it is inherent in many instances there can be no doubt. In insanity, as in other maladies, a great many facts are manifest which we are unable to interpret without an assumption of predisposition or heredity. Why should one member of a family die of phthisis, and another, placed in the same conditions, be unaffected? Schroeder van der Kolk has conclusively proved how insanity and phthisis pulmonalis alternate in the same individual. Or, in scarlatina, independently of its contagious elements, why is it that one child, though unprotected, may be exposed with impunity, and another will contract the malady in its malignant form and thereby perish? Or, again, why is it that after vaccination one person may never again be susceptible to the virus, and another will be re-vaccinated almost at pleasure and vaccinia readily reproduced? In re-vaccinating persons in large communities, I have several times been struck by the fact that the operation was often successful in those who had been inoculated "in the old country" or were distinctly pitted with pock-marks from varioloid. Why should this be? It is probably on account of a marked predisposition existing in the system. Why should one person be effectually protected by one attack of small-pox and another die of a *third* attack, as happened in London in a case

related by Dr. Watson? Such an occurrence must undoubtedly be referred to variable predisposition. So it is in insanity. Some individuals are very much predisposed to it, and labor under a highly unstable condition of the nervous system. In many cases, such being the condition, the patient goes mad, while another person, subjected to exactly the same influences and conditions, will not experience the slightest variation in the performance of his intellectual functions.

Upon inquiry into the history of the insane, you will generally find that the predisposition does not originate with the individual, but existed prior to himself; that is, it is mostly hereditary and transmitted, and this tendency to insanity is an heirloom. You often hear insanity spoken of as a mental aberration, lunacy, a condition of *non compos mentis*, etc.; but the comprehensive term *insanity* is the best of all. Insanity literally means deprivation of reason,—deviation from mental health,—unsoundness of mind. Now, is it practicable to formulate an exact definition of insanity? We will more particularly answer this question in our next lecture, when we revert to this subject. Can a line between insanity and reason be drawn so sharply that all the phenomena found on one side shall be compatible with reason, and those on the other with insanity? This is impossible, and, as a great author says, "The shades of variation in eccentricity, between sanity and insanity, are so slight and numerous that it is exceedingly difficult to state where reason ends and insanity begins." This you might perhaps have had occasion to exemplify, as you may have a friend in whom at times you have noticed very strange actions. Sometimes you will think that if you were not acquainted with the man you would suppose him to be crazy, or not be surprised if some day he should lose his mind. This would be the result of observing that some of his actions were not apparently compatible with the integrity of his mental faculties; while his deportment at other times might entirely remove your apprehensions. Such people may be said to live upon the "*border-land of insanity*," and are very apt occasionally to cross the line and return; occasionally they are seemingly crazy, at other times they are not. Learned, eminent, and practical men "cross the line" sometimes, and make short excursions into the labyrinths of intellectual aberration. At all events, there is in some people a peculiar predisposition to get

over the line, and their peregrinations and rapid transitions mystify medical experts. No matter how much experience you may possess, you will be occasionally unable to determine the status of such cases. Under certain circumstances, fortunately rare, it may take a long time. Months, even, may pass before you will be able to arrive at a definite conclusion, and occasionally you may be baffled after all. This may seem strange at first sight, but after a study of the various phenomena and difficulties of the subject it will not appear so remarkable.

Did you ever see two persons between whom not the slightest difference existed of feature, countenance, or appearance? Even the voice presents a dissimilarity in different persons, being coarse and harsh in some, soft and melodious in others. Understand, that as faces and voices differ, so are mental characteristics distinct. Again, if persons vary so much in their physical and mental capacities and qualities in *health*, how much more will they disagree when affected by *disease*! Besides this, great changes and varieties of development are produced in sane minds under different influences and circumstances. The mental faculties become blunted by neglect of education, or they may be constantly improved and developed by mental gymnastics. In men who habitually study and think, there is, figuratively speaking, considerable mental absorption constantly and automatically occurring, by what Dr. Carpenter terms "*unconscious cerebration*." Some authors contend that the most brilliant thoughts often result from mental activity of this type. Have you never involuntarily thought of something while your attention was engaged on other subjects, and, struck by the pertinency of the thought, immediately noted it, in order to retain it in your memory? Or have you never retired, after assiduously striving to master a perplexing subject, at last abandoning it in despair, to seek repose and to recuperate your energies for renewed efforts, and upon awaking next morning after a sound sleep, found yourself clear-headed and bright, and in perfect possession of what a few hours before you had quite despaired of attaining? This is "*unconscious cerebration*." You were appropriating knowledge unconsciously, and after an invigorating rest the mind manifested what you had thoroughly acquired without perceiving it. It is through the channels of sense that man is educated, by a perception effected in the cerebral convolutions of

whatever the senses may have transmitted; which perception always involves a working of the cortical cells, and in proportion as we cultivate their working power we add to their capacity. As the mind is capable of cultivation, so are the individual senses; and, by the laws of compensation, one sense may supply a lost one, as a blind man's hearing or touch may become preternaturally developed. But suppose a man to have arrived at the age of thirty without having used his senses, and that all at once they be fully developed, what would be the result? He would be an idiot; never having been able to cultivate his senses, he could not appreciate their working, and time must necessarily elapse before he could be enlightened by them. I cannot give a distinct definition of insanity that would be *comprehensive*, because of its protean character, on account of the great and almost infinite dissimilarity normally existing between the minds of different individuals, which will be greatly widened under pathological conditions. Insanity in one person may produce actions not unlike those of the brute, while in another it may lead to actions of a very different character. So that there can be no constant, pathognomonic phenomena which may be said to be attendant upon mental alienation. Of course we have certain classifications, but some of the manifestations of insanity will not admit of any special grouping. I believe that upon the witness-stand you may prudently decline to attempt any definition of insanity, for reasons just mentioned.

"Insanity is," according to Sheppard, "a disease of the neurine batteries of the brain." This definition has at least one merit: it will, he says, "puzzle the lawyers." It sometimes happens that, when medical evidence is required in court, the legal gentlemen, being very shrewd, possessing a knowledge of medical jurisprudence and perhaps some smattering of medical science, seek definitions for their own purposes, knowing how incomplete and unsatisfactory they necessarily are, and will afterwards endeavor to entangle you in their mazes. For these very considerations, the more concise and explicit you are in your testimony, the better. Another definition is that of Maudsley, who states that "insanity is a morbid derangement, generally *chronic*, of the supreme cerebral centres, the gray matter of the convolutions, or the *intelleatorium commune*, giving rise to perverted feeling, defective or

erroneous ideation, and discordant conduct, conjointly or separately, and more or less incapacitating the individual for his due social relations." Now, in some respects this definition is deficient; it is not sufficiently comprehensive, possessing some flaws, and not covering all cases. Still, it has undoubted advantages, because it states—first, where the disease is located, and, secondly, that the disease is usually chronic. According to Van der Kolk, all insanity is acute which has not existed over three months. We are not to infer from Maudsley's reference to the fact of the ailment being usually chronic that it was not originally acute, but simply that a physician is, as a rule, called to treat it after the condition has become more or less chronic. This division of the different results of insanity is also in accordance with the best division of the mind,—that is, *will*, in relation to those parts giving rise to the phenomena of *action*; *feeling*, in relation to those parts giving rise to the phenomena of *emotions*; and *ideation*, in relation to those parts giving rise to *cognition*.

Hence it follows that a man may be insane in his *words*, as evidence of a perverted manifestation of *intellect*; or in his *emotions*, as expressive of a morbid state of those parts which preside over the development of his *feelings*; or in his *actions*, as expressive of disorder of that portion of the brain connected with the phenomena of *volition*; or there may be insanity exhibited in his actions, emotions, and ideas, conjointly; one, or the combination of two, or all the three forms of deranged manifestations just described, may be present.

Man is often said to suffer from moral insanity, which is asserted to be an affective variety, referring exclusively to the development of irregular emotions, as opposed to the perversion of the purely intellectual faculties,—there being in moral, affective, or pathetic disorders a disturbance of the moral faculties only. In former years I advocated this distinction; but of late I have had reason to modify the views I so long taught, and am now convinced that all cases of moral insanity, closely scrutinized, will present evidences of *imperfect ideation*, *superadded to the derangement of the affective faculties*. The point to recollect for the present is, that a person may be insane in one, two, or three forms of manifestation. Sometimes it is difficult to determine the existence of insanity, because some individuals possess a remarkable control over

themselves, perhaps being designing and deceitful ; others, presenting some suspicious symptoms, will talk coherently and intelligently, often puzzling experts as to their actual psychological status. You see, therefore, that a man need not be a raving maniac in order to be insane ; he may be very courteous, intelligent, polished, and affable, and still be hopelessly crazy.

I recollect two interesting cases, which, in this connection, I will relate to you. The first was that of a man subjected to a commission *de lunatico inquirendo*. All the experts who knew him swore that he was insane. But he contended that he knew much more than the doctors did, at least about his own case. He wished to address the jury, was permitted to do so, and made a most brilliant and persuasive speech ; after which, of course, the jury declared that he was not insane, and consequently he was permitted to return home. The very first day of his enjoyment of liberty, he choked his wife nearly to death because she refused to drink out of an old skull which he possessed.

The next case was that of a patient to whose mental alienation I testified most emphatically. He grew very angry with me, and even threatened to horsewhip me. He took exceptions to the fact that the court had appointed an attorney to defend him, stating that he was fully able to protect himself,—that he entertained the most supreme contempt for lawyers ; and at the commencement of the trial he ignominiously dismissed his counsel. He conducted his own case quite energetically and skilfully, examining the witnesses with deliberation and care, seizing the weak points in their answers, evincing great shrewdness and acumen. The jury were evidently staggered by his brilliancy, until one or two preposterous assertions into which he was betrayed convinced every one that his insanity was indubitable. He eventually died of general paresis.

Such cases actually occur ; and these illustrations are not in the least overdrawn. You will often have very cunning insane people to deal with. I remember an old judge who was at St. Vincent's Asylum, and who was a monomaniac on the subject of perpetual motion, though apparently sane on every other subject. He always appeared to be very intelligent, and, after a long stay in the asylum, I felt convinced that he should be discharged. I therefore determined to let him go ; and the judge, exceedingly

thankful and grateful, returned home. Within a week, he became so violent that he was sent to another asylum; and yet I made this mistake after having had him a long time under my immediate observation. So you see that an insane man can successfully conceal his derangement, and, eluding your vigilance, perpetrate a deed of violence. His conversation may show no symptoms of the mental affection, which will sooner or later declare itself. If these sources of error exist, is there any particular rule by which we can clearly ascertain the existence of insanity? Unfortunately, there is not; but there are certain important considerations which have always been of great assistance to me in the *diagnosis* of difficult cases, and which I shall particularly develop in my lecture on "*Emotional Insanity*."

It is strange to what an extent the popular conception of insanity is at variance with fact. In the popular estimation, unless wild, incoherent, violent, or boisterous, one is not insane.

To Folsom, of Boston, a recent author of excellent articles on "Mental Diseases," published in Pepper's "American System of Medicine," I am indebted for much that is pertinent to this lecture. Commenting upon this subject, he observes that, "if maniacal, the timid or frightened young girl who would not hurt a fly, and the tottering, harmless old man, if confused and partly demented, are hurried off to the asylum with the use and show of force suitable for a desperate criminal; while the victim of overwhelming delusions, because he seems clear, logical, and collected, is vigorously defended against the physician's imputation of insanity until he commits an offence against the laws, when he is fortunate if he is not treated as a criminal. It is often impossible for judges, juries, counsel, and even medical experts, to wholly divest themselves of the popular notions of insanity in cases appealing strongly to the passion or prejudice of the day. Cases involving the question of responsibility for crime are decided against science and the evidence, because of certain preconceived notions upon insanity which no amount of skilled opinion can controvert. Jurors, and less often judges, make up their minds what a sane man would do under given conditions, and of what an insane man is capable, judging from the facts within their own experience; and in forming their decisions it is the act itself, and not the man, diseased or otherwise, in connection with the act, that chiefly governs them.

Often they are right, not seldom wrong. Strange, apparently purposeless, illogical, inconsistent action is frequently attributed to the author of it being insane on that subject, whereas he may be simply acting from strong impulse or emotion, and may be by no means insane. On the other hand, because a man knows right from wrong in the abstract, and can ordinarily behave well, the very characteristic workings of his insane mind are often seized upon as unquestionable proof of sanity, even when they admit of no other explanation to the skilled physician than that of insanity."

The above-cited author thinks that the whipping of the insane several centuries ago put an end to much insane conduct, and that in insane asylums of the present day, in spite of the best efforts of the medical staff to the contrary, a brutal, bullying patient is sometimes struck by another patient or an attendant in return for some unusually exasperating and cruel conduct, with the result of making him behave well in future. "It is with reference to this class of cases that the crowd oftenest errs in its definition of insanity. Society claims a voice in the enforcement of laws for its own protection, assuming to know who could control themselves from crime and who not, and naturally wishes the standard of responsibility to be kept high. Of course its sympathies and prejudices largely govern its voice in the matter."

The *legal* conception of insanity is a condition of mind with reference to certain conduct. "An insane man is simply *non compos mentis*. Insanity is irresponsibility." A lawyer's idea of insanity is narrower than that of the physician, regarding it, as he does, with reference to a certain act or series of acts. Folsom holds that in wills and contracts the course is usually clearer than where there is a question of serious crime, and that "*even an insane person in an asylum may be a party to a valid contract*"* or make a will that will hold in law. In this opinion he is in accord with some of the highest authorities, especially with Ray in his "Medical Jurisprudence of Insanity." It is undoubtedly true, as the former authority holds, that a will should fairly represent the wishes and character of the man making it, uninfluenced by insane delusion or prejudice, and that it should bear evidence of a correct appreciation of the circumstances and conditions of the case, and of a

* Italics my own.

mind acting independently, with a reasonable knowledge of the duties involved and of the just rights of others.

"A man is not insane in law unless his act is traceable to, or its nature has been determined by, mental disease affecting his free agency ; in other words, unless insanity caused his act either wholly or in great part." (Folsom.)

The facts should be borne in mind that a very little mental disease can make bad people criminals and may not take others beyond the bounds of propriety. A criminal may become insane and be still pretty much the same kind of a criminal as before.

The question of responsibility is one of the most intricate problems which the physician has to determine. If in such instances the problem were limited to distinctive and well-admitted forms of insanity, no such difficulty would exist ; but everybody knows that only too often the cases, instead of being comparatively simple, are of a most perplexing character. It should never be forgotten that the insane man is just as liable to commit crime as an ordinary criminal, surrounded as he is but too frequently by the same conditions and influences, and incited by the ordinary motives of human action. A man does not cease to be human because he becomes insane. Insanity and humanity are not inseparable nor incompatible. The propensities, motives, schemes, peculiarities, eccentricities, dispositions, moods, and general characteristics of sanity frequently coexist with insanity. The insane as often commit certain crimes as the sane. Truly we may here maintain, with Folsom, that "the evidence is contradictory, the testimony as to previous life and character conflicting, and the disease of so obscure a stage or type that it is almost impossible to form a clear opinion. The determination of a man's degree of free agency is no simple affair which can be decided in all cases by a few or a dozen interviews. Not seldom the mystery remains unsolved after the autopsy. Man's free will is not the property of any substance which can be demonstrated by chemistry, physiology, or microscopical research, but it is the result of the combined action of a whole group of functional activities, the very relations of which to each other are as unknown as their method of action. No stethoscope or ophthalmoscope can reveal its morbid action, which can only be inferred indirectly from the operations of the mind."

The duty of the physician in cases of insanity is,—first, to obtain legal control of an individual's actions, as, for, instance, by the appointment of a guardian,—viz., to institute a *de lunatico inquirendo*; secondly, to deprive the patient of his liberty and place him in an asylum; thirdly, to determine his criminality from a medico-legal point of view, or to estimate his capacity to make a will or contract or to transact business.

It is quite important, therefore, as Folsom continues, "that the medical man should understand that there may be, as regards some particular person, a wide difference between medical insanity, or mental disease, and legal insanity, or irresponsibility. He does most wisely when he confines his testimony to an explanation of the changes caused by disease in the particular case, and to the effect of such changes upon the mind, leaving to the judge's charge and the jury's verdict the questions of guilt and responsibility."

This is excellent advice, and we fully agree with Folsom that this can be the only prerogative of a medical expert, and it is most unwise for him to encroach upon the domain of judge and jury, and pass upon questions of guilt and responsibility, which are entirely outside of the province of a scientific medical witness. No medical expert should ever prostitute his calling by having any interest, financial or otherwise, involved in the questions at issue; nor should he be in any respect biassed in his opinions, either before, during, or after the trial.

Science is truth, and the medical expert upon the witness-stand is there placed "to tell the truth, the whole truth, and nothing but the truth."

It is true that "the laborer is worthy of his hire;" but a physician should not accept either a contingent fee, or one which has any relationship to the interests of either side of the case. His position is a false one if he places himself in the attitude of an advocate, as he is interested solely in supporting and explaining the unvarnished facts, as they are presented to his investigation and analysis by those whose duty it is to arrange them in their correct order and relationship.

In estimating change of character, the individual is to be strictly compared with *himself* at some previous time, and not with some ideal standard of mental health which never existed. Truly, as

Folsom observes, "if we could measure nicely, no two of us could be fairly held to the same degree of accountability."

The questions of the knowledge of the distinctions between *right and wrong*, and of the presence or absence of *delusions*, as bearing upon the general diagnosis of insanity, will be fully discussed in our lecture upon "Emotional Insanity."

Folsom is well sustained in his opinion by authorities when he asserts that "the degree to which the individual deviates from the path of the law may depend more upon his *training and surroundings* than upon his *disease*,—points which must always be considered in establishing a definition of insanity in obscure cases. Of two persons whose circumstances in life, in connection with a certain amount of disease, have produced as nearly as possible identical morbid states, it now and then happens that the necessary surroundings of the one steady and support him, while the associations and conditions of life throw the other still more off his balance. The one is able to sustain the ordinary relations with the world; the other is not."

The *prevention* of insanity is one of the most important duties of the physician; when this is impossible, its early and energetic treatment should engage his prompt and unswerving attention.

This fact will be all the more appreciated when it is recollected how favorable is the prognosis and how successful the treatment of this dread malady in its earlier stages. Again we agree with Folsom, who emphatically states that it is not the doctor's province to punish for crime, but to treat for disease, and that the doctor often fails to appreciate this distinction.

Medical definitions of insanity, remarks Folsom, in text-books and on the witness-stand, do not clearly enough state how far the *medical* and how far the *forensic* meaning of the word "insanity" are implied. What seem to be wide differences of opinion regarding responsibility for crime, as given in the courts, are often due to different ways of stating the question, and nothing more.

Again he observes, "It is impossible to give a satisfactory definition of insanity, to draw any hard and fast line on one side of which we should put all the sane and on the other all the insane. It is not possible to divide insanity from sanity by a single criterion."

Boileau said that all men are insane, the only difference between

them being the varying degrees of skill with which they are able to conceal the *crack*.

Montesquieu observes that insane asylums are built in order that the outside world may believe itself sane.

In 1832, Haslam, one of the first experts in mental diseases in England at that time, testified in court that he had never seen a sane man in his whole life, adding, "I presume the Deity is of sound mind, and He alone."

Savage, in a recent excellent treatise on "Insanity and Allied Neuroses," remarks, "The first question naturally is, What is meant by insanity? I shall try to show clearly throughout this work that no standard of insanity, as fixed by nature, can, under any circumstances, be considered definitely to exist. 'Sanity' and 'insanity,' as recognized by the doctor, and in fact by the general public, must be but terms of convenience. No person is perfectly sane in all his mental faculties, any more than he is perfectly healthy in body."

With due respect to these eminent authorities, I may briefly qualify such opinions as needlessly hyperbolic and impractical.

Spitzka more nearly approaches the truth in this connection when he says, "It may be safely asserted that, in the present state of our knowledge, it is impossible to frame a definition of insanity which, while it meets the practical every-day requirements, is constructed on *scientific* principles. The failure of the best authorities to furnish such a one proves that, until the material elements of mental derangement become more accessible to observation than they now are, scientific definitions must in large part rest on hypotheses. The *practical* need, however, is for a definition which shall include neither ambiguous nor theoretical terms. That the brain is the organ of the mind is an axiom of physiology; that insanity is a manifestation of brain-disorder is a resulting dogma of medical psychology; but, even if we could establish the existence of a brain-lesion in every case of brain-disturbance, we would not be able to formulate the topographical and patho-histological conditions which determine the falling of its manifestations within the boundaries of insanity in one case and without them in another. And neither the axioms of physiology nor the dogmas of medical psychology are regarded with sufficient respect in our courts of law—where the problem of an accurate definition of insanity is

apt to be most emphatically presented to the medical mind—to render their use in filling this gap in our knowledge either satisfactory or profitable.”

In a foot-note, Spitzka adds that “it is significant, in this connection, that none of the most recent German writers on insanity attempt to give a definition of insanity.”

Again, with Blandford, speaking of insanity, we may ask, “Can we even define it?”

“To define true madness, what is it but to be nothing else but mad?”

In truth, its inscrutable appearance without assignable cause in a man hitherto sane, and its no less inscrutable departure, are things which we must confess are not yet explicable by human knowledge.

With Clouston, we emphatically assert that “the whole conduct of things in the world is necessarily so based on the assumption that every man is a responsible being, with a sound mind, that any exception to this, when it occurs, has a very startling effect.”

Baillarger comes very near the whole truth when he states that “*the essential element of insanity is loss of free will.*”*

Ball, of Paris, describes “an insane man as one who, in consequence of a profound disturbance of the intellectual faculties, has lost more or less completely his free will (*liberté morale*), and has ceased thereby to be responsible to society for his actions.”

Krafft-Ebing says that “it is a logical, self-evident proposition that the organ whose function under normal condition is to bring about all mental processes, must be the seat of changes when these functions are disturbed;” and Schüle adds that “the study of disturbances of the mind involves the changes of normal mental functions produced by disease. . . . Mental diseases are brain-diseases, but they are more than that.”

Lord Bramwell once said that “insanity is strong but not conclusive evidence of innocence;” and Lord Blackburn has stated that “the jury must decide in each individual case whether the disease of the mind or the criminal will was the cause of crime.”

Mandsley, in his classic work on “Body and Mind,” remarks, “On all hands it is admitted that the manifestations of mind take

* Italics my own.

place through the nervous system, and that its derangements are the result of nervous diseases, amenable to the same method of investigation as other nervous diseases. Insanity has accordingly become a strictly medical study, and its treatment a branch of medical practice. Still, it is also too true that, notwithstanding we know much and are day by day learning more of the physiology of the nervous system, we are only on the threshold of the study of it as an instrument subserving mental function. We know little more positively than that it has such function ; we know nothing whatever of the physics and of the chemistry of thought."

LECTURE XIII.

INSANITY—*continued.*

Classifications—Maudsley's Classification—Etiology—Predisposing Causes: Climate, Religion, Civilization, Sex, Period of Life, Deficient Education, Individual Predisposition, Insane Temperament—"Border-Land of Insanity"—Exciting Causes: Masturbation, Drunkenness, Epilepsy, Transmutation of Nervous Diseases, Chronic Diseases, Disorders of the Sexual Functions, Injuries to the Head—Moral Causes.

GENTLEMEN,—In my last lecture, when speaking of insanity, I referred to important facts regarding its character and definition. It now remains for me to *classify* this disease in accordance with the views of the best authorities. It is quite as difficult a matter to classify insanity as to define it, and there are instances where it is impracticable to arrange some of its manifestations typically.

I am a believer in the simplest possible division of the different varieties of this disease, in order not to overburden the mind, and am convinced that Pinel's method is most satisfactory, being as follows: 1st, *Mania*; 2d, *Melancholia*; 3d, *Dementia*; 4th, *Imbecility* and *Idiocy*. (The last two affections are considered jointly simply for practical purposes.) Authors have studied the subject under almost numberless classifications. Dr. Skae has originated a very elaborate system, whose probable advantage consists in the arrangement of the different types according to their *etiology*.

Van der Kolk, of Utrecht, to whom medical science owes a great debt, retains two principal forms: 1st, *Idiopathic insanity*, arising without any appreciable cause, primary in character, and originating exclusively in the brain. (In my last lecture I explained that insanity is always located in the brain, though sometimes its origin may be from sources altogether foreign to that organ.) 2d, *Sympathetic insanity*, which includes all forms proceeding from different portions of the human economy, as, for instance, the viscera of the thorax, or, more frequently, of the abdomen or the pelvis.

You will thus perceive that the origin of sympathetic insanity may be sought for in the heart and lungs, in the liver, spleen, small intestines, colon, etc. *Insanity is indeed quite often sympathetic with a diseased colon*,—especially where *melancholia* exists,—the starting-point not infrequently being in some portion of the alimentary canal. Again,—as all practitioners very well know,—certain diseases of the genito-urinary apparatus not rarely produce the most obstinate forms of mental aberration. It is not difficult to understand why diseases of these organs should sometimes have this determination, if you take into consideration the complete harmony which normally exists between all parts of the body and the central organ of the nervous system,—the brain. No matter how remote their situation, many organic complications give rise to mental disturbances by the *reflex* irritation excited first in their nerve-fibres and then propagated to the central nervous dépôt.

One more fact in connection with the sympathetic form is, that these remote causes, though primarily exciting only simple functional disturbances of the brain, will, if not removed,—the reflex irritation persistently continuing,—produce organic disease, developing an incurable form of insanity. The more obstinate the duration and the more violent the irritation, the greater is the danger of organic changes in the histological elements of the brain,—the resulting insanity becoming irremediable.

The classification I have adopted is that of Maudsley, and is as follows :

“If a broad division were made of insanity into two classes,—namely, insanity without positive delusion and insanity with delusion,—in other words, into *affective* insanity and *ideational* insanity; and if the subdivisions of these into varieties were subsequently made,—would not the classification, general as it may appear, and provisional as it should be deemed, be really more scientific than one which, by postulating an exactness that does not exist, is a positive hinderance to an advance in knowledge? One desirable result of great practical consequence could not fail to follow,—that is, the adequate recognition of those serious forms of mental derangement in which there are no delusions. I have ventured accordingly in a former publication to put forward the following classification :

"I. AFFECTIVE OR PATHETIC INSANITY.

1. *Maniacal perversion of the affective life. Mania sine delirio.*
2. *Melancholic depression without delusion. Simple melancholia.*
3. *Moral alienation proper.* Approaching this, but not reaching the degree of positive insanity, is the *insane temperament.*

"II. IDEATIONAL INSANITY.

1. *General.*
 - a. *Mania.*
 - b. *Melancholia.*
2. *Partial.*
 - a. *Monomania.*
 - b. *Melancholia.*
3. *Dementia*, primary and secondary.
4. *General paralysis.*
5. *Idiocy*, including *imbecility.*"

The next time we meet I shall give a short description of these forms. Contenting ourselves for the present with this brief reference to the classification adopted, we next shall consider the "*border-land of insanity.*"

A few words concerning this "*border-land.*" It is of great importance to admit the existence of a border-land between sanity and insanity, and to investigate carefully the peculiarities of the cases that exist therein.

Maudsley, in commenting upon this subject, remarks, "Assuredly it is a fact of experience that there are many persons who, without being insane, exhibit peculiarities of thought, feeling, and character which render them unlike ordinary beings and make them objects of remark among their fellows. They may or may not become actually insane, but they spring from families in which insanity or other nervous disease exists, and they bear in their temperament the marks of their peculiar heritage. They have, in fact, a distinct neurotic temperament, a certain *neurosis*, and some of them a more specially insane temperament, an *insane neurosis.*"

In treating of the connection between crime and insanity, the same writer, in his work on "Responsibility in Mental Diseases," speaks as follows: "If the secrets of their natures were laid open, how many perverse and wrong-headed persons, whose lives have been a calamity to themselves and others, how many of the depraved characters in history, whose careers have been a cruel chastisement to mankind, would be found to have owed their fates to some morbid predisposition!"

Regarding the influence of physical effects upon temperament and moral well-being, Dr. Wigan observed, "I firmly believe that I have more than once changed the moral character of a boy by leeches to the inside of the nose."

Dr. Savage, in his work on insanity, says, "There can be no doubt in the minds of those who see much of the criminal classes and of those who see much of the insane classes, that there is something in common between them. I must not be misunderstood in saying this, for there is an immense difference between some insane persons and some criminals; but, as the savage and the statesman have connecting links between them, so among criminals and lunatics there are many grades which approach one another very closely. To begin with, the physical aspect of a chronic lunatic resembles very closely that of a confirmed criminal, and the mental degeneration of man leaves his features so changed and debased that he resembles the man who, from vice of birth or faulty surroundings, has never developed the higher social qualities. As Dr. Maudsley has well pointed out, 'Though there is a *border-land*, there is no boundary-stone; and there are cases in which exist some insanity and much crime, and others with much insanity and little crime.' And at present this *border-land* is the one on which most forensic battles have to be fought. Though from theoretical points of view it may appear that any one with criminal tendencies must be looked upon as insane (as one, in fact, who cannot be calculated upon, and whose actions are not governed and controlled as are those of the ordinary social unit), yet society, as represented by lawyer, judge, and jury, will continue to hold the balance in its own hands, and punish those who may, after all, be of unsound mind, the expert's opinion notwithstanding."

ETIOLOGY OF INSANITY.

Insanity is not, as was formerly supposed, a mere hypothetical disease,—an imaginary or metaphysical affection of the mind. The mind, being intangible and immaterial, cannot be subjected to disease. Insanity is a disorder of the *brain*, just as pneumonitis is an inflammation of the lungs, pleuritis an inflammation of the pleural sac, or typhoid fever a specific blood-poisoning; and the disordered evolution of mental phenomena observed during the course of insanity is but the morbid manifestation of a brain which, from physical causes originating in disease, is unable to perform its proper physiological functions.

The fact that insanity is a disease of the brain is one of the greatest importance, showing us the gross injustice inflicted in times past upon individuals, casting upon them, as it did, the stigma of social ostracism. It was supposed that they were suffering from some uncommon malady wrought by mysterious influences, or sent as a punishment from the gods. Such theories were as preposterous as they were unfortunate. All individuals are more or less liable to this calamity. But why are some people insane and others not? Why does insanity not affect indiscriminately all the members of a family? Why does it show a predilection for certain persons, selecting its victims with remarkable consistency? We have already adverted to the laws relating to this liability, and have considered it as founded in an "*unstable condition of the nervous elements*," peculiarly prone to perturbation of their equilibrium, whereby more or less interference with all the functions connected with the development of mental phenomena is produced, the person thus suffering ceasing to be rational and responsible. Where such a predisposition exists there is likewise an unstable constitution of the cells connected with the evolution of the moral or intellectual faculties; and this instability is born with the individual and inherent in him, having existed from the first moment of his life. Let us suppose two individuals, one of whom is subjected to violent mental emotions, or to other causes eminently calculated to lead to insanity. Should no predisposition exist, this person will almost certainly pass through the ordeal unscathed. The second individual, on the other hand, possessing the liability, on being exposed to precisely the same

agencies, will most probably become mad,—the cortical cells now no longer performing their duty normally.

If we knew all the causes leading to insanity, or could foresee contingencies which may arise, then we might venture to predict insanity in certain individuals or families at particular periods of their lives. However, insanity does not depend upon one, or a few, but upon a *multiplicity of causes*; and, as Maudsley remarks, “hereditary, predisposing moral and physical causes are not alone necessary, but a combination, a concurrence of conditions, and then lunacy follows.”

Let us now review some of the leading causes of insanity.

1st. *Climate*—which seems to have some influence in the production of this malady. Indeed, I have frequently observed that on dark, moist, gloomy days the majority of insane persons are worse: the melancholic cases are more deeply depressed, and the maniacal are more difficult to control. There seems to be also a peculiar disposition towards a cure at certain periods of the year,—in spring especially, when everything in nature is being endowed with renewed life and vigor. In curable patients we frequently find the disease singularly yielding at this time of the year,—a fact mentioned by Esquirol. Most of you have probably experienced a peculiarly depressing effect upon a gloomy day when the sky is overcast. And if this influence be felt by sane persons, how much more will it impress the insane, or those in whom an hereditary predisposition exists! Should this influence be unduly continued in such predisposed persons, insanity may be developed; a fact which partly explains why suicides are so frequently committed during dull and dreary weather.

2d. *Religion*, it is held, is often a cause of insanity. In nervous temperaments subjected to *intense* religious excitement, as *revivals*, for instance, danger of mental disease is to be apprehended. I have witnessed many instances of this kind, disastrous nervous complications having fanaticism as their immediate precursor. Indeed, the effects of the mental emotions, reacting upon the physical organism, are so extraordinary, owing to their *expansive* character, that it is easy enough to understand how they may be fruitful sources of insanity. Maudsley aptly remarks, in his work on “Body and Mind,” that “when the emotions are very much disturbed there is a proportionate disarrangement in the

cortical cells presiding over the moral functions, nervous influence being liberated with great force ; this is transmitted along certain appropriate nerves, and the impulse consequent upon the original liberation of nerve-force may be so great as to produce intense and notable effects at distant points, where it is received and ultimately distributed." These phenomena may be compared to those produced by a powerful galvanic battery generating a current of great tension transmitted along conducting wires, a violent shock occurring at the point of arrest ; while if this point of arrestation furnish a resistance corresponding with the strength of the discharge, destructive and disorganizing effects will ensue. This is not unlike what occurs when a person is struck by lightning. Agitation from either intense grief or unbounded joy may be so excessive as to produce sudden death. Death thus occasioned was formerly commonly supposed to be the result of a broken heart ; but in reality it is simply the effect of the overpowering violence of the emotions upon the nervous centres, and not, as was previously imagined, a muscular laceration.

3d. Another predisposing cause is said to be *civilization and its progress*. It is true that among savages insanity is exceedingly rare. In what way does civilization tend to produce insanity ? you ask. Perhaps by the expansive wants therefrom arising, often to be satisfied only at the expense of health,—all the powers of man's organization being brought into requisition and overtaxed in the daily struggle for self-maintenance and individual pre-eminence. In many instances where labor is chiefly of a mental character, there is but too often an excessive strain upon the intellectual faculties. Should this happen in connection with hereditary predisposition to insanity, it is probable the mind will succumb, its possessor being thrown back among the crowded ranks of the unsuccessful, and sunk perhaps into hopeless lunacy. Indeed, in the professional man, whose bread is usually entirely dependent upon never-ceasing brain-work, the retrograde metamorphosis of tissue must be enormous ; and should the slightest instability in his nervous system exist, the sum of his efforts may eventually cause a disturbance leading to the development of insanity. In this way may civilization be productive of the disease. It is estimated that in civilized communities the proportion afflicted is about one in five hundred.

4th. *Sex* undoubtedly exercises some effect ; for instance, a nervous, hysterical woman is much more liable to become insane than a delicately-organized man exposed to the same influences. In debilitated women, especially in moments of joy or grief, you always have cause to dread incidental insanity. When a mother loses her darling child, the emotional disturbances are often terrible, and you may fear the result ; but these apprehensions naturally do not extend to the father. Indeed, it is very improbable that a man could be constituted with such a delicate, nervous, and excitable temperament as a woman : his very organization precludes this idea ; and it is doubtful if he be capable of experiencing the same intensity and exaltation of the emotions as his more frail and tender partner.

5th. The *period of life*, also, seems to possess a certain degree of influence as a predisposing cause of insanity. At the time of puberty, and also among women at the appearance of their menopause, we often find the development of mental complications. In man, even, it is contended that an equivalent *change of life* occurs at a certain advanced age, though not always accompanied by a loss of sexual capacity, the sexual proclivities frequently seeming to be enhanced, as a prelude to approaching decay. At this time, in consequence of the activity of the retrograde processes, insanity seems markedly to increase.

6th. *Education* is the next cause to be considered. In deficiently-educated children (I refer principally to *moral* education), whose training has been neglected, whose propensities to evil have been gratified rather than checked, whose bad temper has been developed instead of restrained, and in whom any instability of the nervous element exists, the least exciting cause may bring about insanity. It is a mother's care and training which make us the men we are ; and we can never sufficiently appreciate her tender discipline and solicitude, since but for her constant and untiring efforts we might have been perverse, if not insane. On the other hand, excessive mental strain, such as is sometimes required in the school education of our youth, may be equally pernicious.

7th. *Individual predisposition* is a cause to which I have already adverted. It is consequent upon hereditary tendency, or it may be acquired. Many authors state that at least fifty per cent. of

the cases of insanity are of ancestral transmission ; and I am inclined to the belief that even this is an under-estimate. It is also asserted that this inheritance is more apt to descend from the mother than from the father. Children are in much greater danger when their *mother* has been insane prior to their birth than when the disease appears at a subsequent period. Insanity is perhaps more readily transmitted to daughters than to sons.

8th. The *insane temperament* is that condition in which "an individual is, by reason of a bad descent, born with a predisposition to insanity ; he has a native constitution of nervous element which, whatever name we give it, is unstable and defective, rendering him unequal to the severe stress of adverse events. In other words, the man has the insane temperament ; he is liable to whimsical caprices of thought and feeling ; and although he may act calmly and rationally for the most part, yet now and then his unconscious nature, overpowering and surprising him, instigates eccentric or extravagant actions ; while an extraordinary and trying emergency may upset his stability entirely." (Maudsley.) "He suffers from the worst of all tyrannies, the tyranny of a bad organization." (Sheppard.)

This mental condition is most important in its medico-legal bearings ; and the defence of Joseph Fore—who was tried for murder in the first degree and acquitted—was conducted successfully upon this very theory. His subsequent actions and behavior and sad death clearly substantiated the fact that the plea was well grounded and justly maintained.

The *exciting causes* of insanity are divided into *physical* and *moral*.

1st. A very frequent physical cause of insanity is *masturbation*, a disgusting vice, to which, I must say, I believe an astonishing number of lunatics, either as a cause or as a consequence of their disease, are victims,—many more than the statistics of asylums show, owing probably to the false delicacy which causes patients and their friends to conceal the truth.

2d. *Drunkenness* is another of the causes of insanity. Men are often habitual drunkards before they realize it ; they then find it more difficult to dispense with liquor than with their meat and bread. Experience leads me to believe that inebriety as an exciting

cause constitutes the principal factor in about one-third, if not in one-half, of the whole number of cases of insanity.

3d. *Epilepsy* is mentioned as a cause of insanity by some authors, while others contend that it is more frequently a result thereof. This we shall attempt to decide farther on.

4th. *Transmutation of nervous diseases*, so called by Trousseau, meaning that a nervous disease in one generation may be transmitted to the next or the second generation, but with a change in form, is also a cause of insanity. Hence we observe chorea in the children or grandchildren of epileptics, and *vice versa*, these diseases not infrequently resulting in insanity. It sometimes happens that the transmission of nervous disease will take a different form for each member of a family,—neuralgia in one, alcoholism in another, epilepsy or hysteria in a third, and in a fourth, insanity.

5th. That *chronic diseases* are remarkably productive causes of insanity is not an astonishing fact when you recall what I have stated as regards the necessity of a normal state of the blood in order to insure a healthy condition of the functions of the nervous system. Consequently, an anæmic or a hyperæmic brain is incapacitated for a proper performance of ideation. The same result would happen in rheumatic or gouty affections, in syphilis and other diseases due to blood-poisoning, whether of a vegetable, an animal, or a mineral character, or whether the result of dyscrasiæ or of acute febrile diseases. Insanity, however, is by no means an inevitable result in all cases of this nature,—the absence or presence of an inherent predisposition playing the most important part in its superinduction. There is, moreover, as I have told you, a certain class of patients who do not remain permanently insane, but after an attack of insanity return to their normal condition, *to relapse again*, making it very difficult to anticipate their future status with any certainty.

6th. *Derangement of the sexual functions* has an extraordinary relation to insanity. In females especially many abnormal conditions of the uterus, such as retroversion, prolapsus, etc., are active agents in this respect. In curing the primary affection you will cause the entire disappearance of the insanity.

Van der Kolk relates a case of prolapsus, attended by melancholia, which was relieved upon the reduction of the prolapsus. But on removing the pessary the symptoms of melancholia imme-

diately reappeared, to vanish again upon renewed reduction. This statement admits of no doubt whatever, having been confirmed by the experience of numerous other alienists. "It is certain that an attack of mania has followed the suppression of the menses, and that the return of menstruation is often followed by recovery from insanity; but it is certain also that outbreaks of maniacal fury, or of suicidal or of homicidal violence, have coincided with the period of menstruation. . . . Fleming relates two cases in which melancholia was cured by the use of a pessary, in one of them regularly returning whenever the pessary was removed; and I have seen, in one case, severe melancholia of two years' duration disappear after the cure of a prolapsus uteri. Instances are on record in which women have regularly become insane during each pregnancy; and, on the other hand, Guislain and Griesinger mention a case, respectively, in which insanity disappeared during pregnancy, the patient at that time only being rational." (Maudsley.) Some of these women become insane before parturition, some immediately after, and others during lactation. These facts are of importance, and, whether a specialist or not, the physician cannot afford to ignore them, as their significance is evident.

7th. *Injury to the head* is often the cause of most insidious, dangerous, and intractable forms of insanity. I believe that all severe blows upon the head, sooner or later, may cause very serious brain-symptoms, though years may elapse before their appearance. In such cases, if you carefully analyze their history, it will be evident that the insanity was, beyond doubt, directly caused by the blow. Forbes Winslow contends that these disastrous and insidious consequences resulting from injuries of the head are too often overlooked. I have witnessed a case of pachymeningitis and resulting cerebral abscess from extension of the morbid process by contiguity of tissue, the effect of a traumatic injury of the cranial vault inflicted twenty-three years previously. In the mean time the individual had amassed a large fortune by industry and successful financiering.

The *moral causes* of insanity consist simply in undue violence of the emotions, which, when occurring in persons possessing an inherent tendency to insanity, very seldom fails to result unfortunately. Among the more dangerous emotions we find those of anger, jealousy, hatred, and love very prominent. Sudden reverses

of fortune, and the consequent sensations, have often caused a dethronement of reason. Not only a change from wealth to poverty, but the reverse also may result in mental disease. The excitements incident to war, and its disastrous consequences, have afforded ample illustrations of insanity consequent upon violence of the emotions. In a word, whenever the mind is subjected to undue strain, or whenever the passions are extraordinarily roused, other conditions being favorable, mental alienation may ensue.

LECTURE XIV.

EMOTIONAL INSANITY, AND ITS MEDICO-LEGAL RELATIONS.

GENTLEMEN,—Insanity is necessarily a disease that must interest you all, because it is a physical affliction which sooner or later may invade the sacred precincts of your own homes. It is a disease which, in the present condition of society, is becoming extremely rife, keeping pace, some authors hold, with advancing civilization.

A certain fascination clings to the study of mental pathology. Clinical observations teach us that the wear and tear, the stress, the contentions, troubles, competitions, and annoyances of every-day life, engulf many minds, so constituted, in consequence of inherent weaknesses, as not to be able to resist such inevitable influences. In other words, as men must bear the strain and friction of every-day life, insanity correspondingly increases.

In the present period of pathological enlightenment, the fact that insanity is a disease of the brain should admit of no dispute. The interest of its study will be enhanced by our natural efforts to unravel its multitudinous phenomena; and in seeking for explanations of its complex manifestations we are often compelled to attempt to explore the labyrinths and mysteries of brain-action. Of the latter, it is true, we have overmuch to learn; nevertheless, the researches of modern science have not been entirely fruitless, but have been productive of excellent, if not indeed very remarkable, results.

We can at least, in consequence of this progress in the right direction, dismiss the old mythical theories of the metaphysicians,—obsolete views, never again to be rejuvenated,—which taught people to believe that in dealing with the phenomena of insanity they were witnessing or combating nonentities, shadows, spiritual processes, mystical conditions of still obscurer origin, as opposed to the purely physical symptoms, the outcroppings or manifesta-

tions of brain-lesions, more or less demonstrable in all cases when subjected to the crucible of modern investigation.

Formerly insanity involved social ostracism, and the prejudices which its supposed stigma originated exist with curious pertinacity even at the present day, notwithstanding the teachings of science. It was once considered necessary that the victim of this disease should forever be sequestered from society, and his misfortune involved his family in almost equal disgrace.

We know now that insanity is simply a physical affliction, instead of a *morbus sacer*, and the result of certain well-recognized pathological deviations; that it not infrequently originates in congestions and inflammatory conditions of the brain; and that, just as pneumonia is an inflammation of the lungs, pleuritis an inflammation of the pleura, and nephritis an inflammation of the kidneys, so is insanity a departure from the physiological status of the brain necessary for the proper evolution of healthy mental and emotional phenomena, having its starting-point in the same vascular and irritative disturbances that give rise to other pathological states.

Van der Kolk divided insanity into idiopathic and sympathetic insanity. The former originates primarily in the brain proper, the latter is but the reflex radiation of remote disturbances, starting frequently in other parts of the human organism, and propagated in constant irritative waves towards the central organ of the nervous system. Diseases of the viscera, whether situated in the pelvic, the thoracic, or the abdominal cavity, are common causes of insanity, which in such instances is superinduced by the reflection of such disturbances upon the brain, resulting in congestions and inflammatory conditions, sooner or later inducing insane manifestations.

Such a result is not astonishing when the wonderful sympathies of the brain with every portion of the human economy are taken into consideration. The intimate relation of the brain with every other organ, blending various interests in one harmonious whole for the common weal, is an indisputable fact.

This is still less difficult to appreciate when it is recollected that the brain is the supreme centre presiding over all other parts of the nervous system and the animal economy. Indeed, we can hardly conceive of any constituent atom of the body, no matter

where found, which is not in more or less intimate relation with the brain. Hence may it well be said, as has been done by a certain writer, that when a man becomes insane he is insane to the very tips of his fingers.

The brain may truly be said to be the seat of the regulative force of all the phenomena of the mind, of the emotions, intellectual acts, and volitional manifestations.

The term "insanity" is comprehensive in character. When we speak of insanity in general, we do not refer to one particular form of the disease, but we include in the term a very great variety of morbid brain-manifestations. Just as the word "tumor" has frequently the most varied significance, suggestive, as it is, of many physical varieties and characteristics, numerous classifications, terminations, and pathological origins, so the word "insanity" is to the psychological physician a mine so rich and varied in its productions, so deep, intricate, and labyrinthic, as to be quite inexplorable to the limit we desire. You can therefore form some adequate idea of the comprehensiveness of this term "insanity."

Before studying emotional insanity in particular, let us consider some other points connected with the general subject of insanity.

Is there anything in the aspect or appearance of the insane which will enable us invariably to recognize the existence of the disease by a casual observation of the physiognomy? A fallacy of very common occurrence exists in this connection.

During medico-legal contests, we often hear allusions by the contesting lawyers to the "wild eye of the insane," as a proof of incontestable alienation. This method of recognition of the disease rests upon a very unscientific basis.

All physicians who are familiar with the phenomena of insanity, and are accustomed to explore "the mind diseased," in judging of the presence or absence of the former, know full well the difficulty and fallibility of conclusions based upon such premises.

A careful study and analysis of the physical conditions or the symptomatic manifestations of insanity will clearly indicate how little can be determined by attention to the countenance alone. Let any person who doubts this assertion visit the wards of a lunatic asylum, and, after a purely demented patient has been observed (whose features unmistakably indicate the departure of

all reason and expression), let him watch the large majority of patients who remain, engaged, as they may be, in billiards, chess, the perusal of periodicals, and the enjoyment of cards, and, while giving an attentive ear to their conversation and studying their features, endeavor to determine from such observation alone whether or not reason be dethroned. His task would be one of herculean magnitude. It must not be imagined that insanity is an affection whose presence we can affirm in our patients without most careful scientific analysis and scrutiny. "Some of them understand the nature of their disease quite well, discuss their cases intelligently, and frequently ask why they, automaton-like, are impelled by a force which they cannot resist to constantly do things which their intelligence and better nature condemn. Not a few are confined in places of safety by their own preference." (Folsom.)

It is not to be supposed that medical experts experience convictions as to the presence or absence of mental aberration without careful subjection of the data which should mould their opinions to the laws of diagnosis governing a recognition of all other diseases. Without such a procedure their investigations would be unworthy of scientific men.

With a careful adherence to such methods of observation, based upon broad principles of medical science, the deductions from certain premises will be susceptible of proof and remain established upon an immovable basis. Just as in the diagnosis of pneumonia auscultation and percussion are practised by the prudent physician as aids in evaluating the rational symptoms, so in the diagnosis of emotional insanity all the facts, including an attentive study of all collateral phenomena, must be collectively weighed. What, then, are the logical deductions upon which we may predicate an opinion of this form of insanity?

1. *Hereditary predisposition* is one of the many important links of the pathological chain of sequences which has to be developed in the diagnosis, and is one to which the physician should first direct his attention. Ancestral taint, the fact that the parentage of the particular person undergoing investigation is tinged with previously existing insanity or allied neurotic affections, must never be lost sight of. Persons having such an unfortunate inheritance are the victims of a constitutional instability of

nervous equilibrium which the most trivial circumstance may disturb. That it is inherent in most instances, there can be no doubt.

In emotional insanity, as in other maladies, a great many facts are manifest which we would not be able to interpret but for the assumption of predisposition or hereditary transmission.

In many conditions of life numerous people become insane, while others subjected to exactly the same influences will experience no deviation from their normal mental health. This tendency to insanity is, therefore, but too frequent an heirloom.

2. *Causation* is the next point for consideration in our diagnosis of emotional insanity. *Predisposing causes*, including sex, period of life, condition of life, individual predisposition, education, and many other conditions, must be studied.

Exciting causes, subdivided into *physical* and *moral*, next demand attention. Relegated to the former are such influences as intemperance, masturbation, epilepsy, chronic diseases which deteriorate the blood, which latter conditions are prolific of insanity, exemplifying Cicero's well-known maxim, *mens sana in corpore sano*. Then, again, injuries of the head, lactation, pregnancy, uterine diseases or disturbances, puberty, the climacteric period, all are of the utmost significance in an etiological sense.

Of the moral causes Maudsley asserts that "it is not in the way of great intellectual exercise, when unaccompanied by emotion, to lead to mental derangement; it is when the feelings are deeply engaged, when the mind is the theatre of great passion, that it is most moved and its stability most endangered."

Anxiety, jealousy, sorrow, disappointed affection or ambition, are powerful factors in this regard. Causation, therefore, plays a most important part in our diagnosis.

But it is more especially a *concurrence* of causes that leads to a development of this form of the disease. If we knew all the causes leading to it, or could foresee contingencies which may arise, then we might venture to predict it in certain individuals or families at particular periods of their lives.

However, as before observed, insanity does not depend upon one, or a few, but upon a *multiplicity of causes*; and, as Maudsley remarks, "hereditary, predisposing moral and physical causes are not alone necessary, but a combination, a concurrence of conditions, and then lunacy follows."

To recapitulate, various potentialities—the existence of disease, acute or chronic, which leads through physical developments to the production of emotional insanity; particular conditions of the organism; all varieties of physical deteriorations; impaired or impeded nutrition; poisoned conditions of the blood; excessive functional activities, however produced; the absence of sleep; reflex actions or sympathies; quantitative as well as qualitative disturbances of the circulation—are energetic factors in the production of mental disease of this as of other varieties requiring the psychological physician's closest scrutiny.

The development of psychic functions and their conformity to a physiological type require a healthy brain for their proper evolution. Diseased brains lead to culminations of morbid mental and emotional manifestations and phenomenal deteriorations known as insanity.

Brains intellectually overworked, emotionally over-excited, like jaded horses tasked too heavily, suffer their functional activities to be overwhelmed, and without sufficient recuperative powers total ruin and wreck inevitably follow.

That moral causes, especially the depressing passions, should prove energetic agents in the production of mental disease, need not excite our astonishment. Moral shock not infrequently overwhelms the entire emotional and physical nature of the individual. That powerful emotions are productive of disease—nay, even death—we all know. Medical literature is stored with facts which prove conclusively that the hair sometimes turns gray in a single night.* Maudsley ("Body and Mind") observes "that a sudden and great mental shock may, like a great physical shock, and perhaps in the same way, paralyze for a time all the bodily and mental functions, or cause *instant death*."

3. "*Change of character, without any adequate external cause,*" is the next link of importance in the diagnostic chain we are forging, without which all our efforts to establish the existence of emotional insanity in a given case would be invalidated. Its importance is pre-eminent. It is the pivot on which the diagnosis turns. This change consists in a departure from one's normal self, not merely a supposititious or hypothetical change, but a

* Tuke, "Influence of the Mind upon the Body."

change established in *propria persona* by the rigid scrutiny of scientific analysis.

Daily actions, sentiments, natural impulses, propensities, passions, moral dispositions, feelings, affections, inclinations, habits, are compared with a *normal standard*, a departure from which must be evident to those whose previous acquaintance and intimate relations with the subject peculiarly adapt them for reaching proper and indisputable conclusions.

Under these circumstances reliance must unavoidably be placed by the physician upon the experience, observations, and inferences of near relatives, who are better qualified to judge of early abnormalities or deviations of the affective life than the most skilful diagnosticians.

A mother, for example, can certainly establish a change of character in her child better than any other person. The physician must, however, satisfy himself as to the absence of all possible motives of action, and of all other external adequate causes, which in the absence of *disease* could sufficiently explain these singularities of conduct, eccentricities of action, and changes in one's natural self, or a departure from that type previously characteristic of the individual.

This departure consists in a great variety of gross or delicate alterations, which it is the duty of the physician to educe. These must so change the person under investigation that he ceases, as it were, to be the same individual that he was. I am making no reference to discoverable intellectual perversions. I am simply considering deviations in the affective life, presented only in an analysis of actions, as expressive of morbid feelings, not of words, as illustrative of disordered ideas. The social, domestic, religious, and business habits and antecedents must be scrutinized with the utmost rigidity and fulness of detail. Heretofore an affectionate husband and tender father, the caresses lavished upon his family cease; he repels the advances of his little flock which before constituted his delight; there is a sad revolution in his home life, and the dear ones of his fireside are the first to recognize the stern reality which confronts them. This recognition of the patient's abnormal condition occurs long prior to any overt act of insanity or explosive violence.

The bursting of the storm is long anticipated, however, by the

keen perception of those whose intimate relations with the patient have caused them to realize its direful foreshadowings. So in all other conditions of life, whether social or domestic, these changes will be perceptible.

Men of strong religious natures and convictions suddenly become rash, intemperate, obscene, desperate, and utterly depraved. Such transitions develop themselves without any reason assignable by the most intimate companions.

An eminent and learned divine, distinguished for social virtue, high and charitable religious aspirations and disinterestedness, the centre of attraction and the adulation of his flock, admired, loved, and respected by all, *suddenly, without external adequate cause*, becomes irreligious, intemperate, profligate, and wretchedly dissipated. If, moreover, without notice, he indulge homicidal or suicidal propensities of *recent* development, we would judge him either a confirmed lunatic or previously a consummate hypocrite; for the study of his life must confirm one or the other hypothesis.

Or suppose a parsimonious man, taking care of every dime, exhibiting a ceaseless and sleepless solicitude for his worldly goods, never giving or expending anything except for the absolute necessities of life, were suddenly, without adequate motive, to become excessively liberal, squandering his money as recklessly as formerly he had carefully hoarded it, would not such conduct point to a most suspicious perversion of feeling, and to a change of sentiment consistent only with emotional insanity? Hence the *significance* of *actions* when contrasted with the previous conditions or antecedents of a patient.

The foregoing illustrations especially indicate, therefore, the development of insanity of the affective type, without any particular concomitant evidences of the existence of morbid intellection.

In business relations, if heretofore one has been honest, upright, reliable, a representative man in his sphere of life, whether commercial or professional, a change so perceptible will manifest itself as to cause his acquaintances to be staggered at its completeness and suddenness. The perturbation of his moral nature will be evident to his most casual acquaintances.

But, gentlemen, think you this is all to be considered in this connection?

When you essay to diagnosticate emotional insanity, you must not only dip deeply into the questions I have already presented for your study, but you must investigate all proved morbid tendencies with equal thoroughness. Suicidal and homicidal propensities are particularly to be examined. When such tendencies—without a motive—are proved, they constitute strong corroborative proof of the existence of insanity.

Such links being necessary to establish the existence of emotional insanity, we can but regret and deprecate opinions that once influenced a jury in a celebrated trial in the State of New York to bring in the absurd verdict that a man was sane one moment prior to the commission of the homicide, insane during the homicide, and sane immediately subsequent to the act.

Such opinions bring science into disrepute and prostitute the plea of insanity, exciting the clamor of popular prejudice when, in the great cause of humanity, mental alienation is sought to be proved in genuine cases.

4. "*Want of harmony of the individual with his surroundings.*" "It is important, therefore, that we have in remembrance the individual's social relations when dealing with moral insanity, as we regard the very different relations of an epithelial cell and a nerve-cell when dealing with structures so far apart in the scale of life. It is chiefly in the degeneration of the social sentiments that the symptoms of moral insanity declare themselves; it is plain that the most typical forms of the disease can only be met with in those who have had some social cultivation."

To establish this is a matter of the utmost importance, for "the morbid phenomena of the diseased mind witness in some measure to the degree of its previous development, yet the degeneration which disease implies must needs display itself in an alteration in the kind of manifestation of *feeling, thinking, and acting*,—in other words, in a changed self,—while, again, the import, as morbid, of the phenomena displayed, can only be rightly weighed in relation to the individual sphere of life. It is, for example, quite possible, though apt to be forgotten in practice, that *sentiments and acts which are habitual in the lowest station of life may be sure signs of mental disease when uttered and done by one in a high social sphere.*"

There is under such circumstances a want of congruity in

social relations, a want of adaptation between the general and particular social harmony of the individual. "A cancer is physiologically illogical; nevertheless it persists, and finally kills the patient, being pathologically logical."

The individual who no longer constitutes a part of the general harmonious whole, for reasons "psychologically accountable," is no longer the unit in the social system which he was prior to the inroads of emotional perversion. "Insanity destroys the relations and responsibilities of the individual in the social system, making him very much like what a morbid element is in the organic system,—something which cannot take its due place in the general harmony, and which must either be eliminated from it or sequestered and rendered harmless in it." "Schopenhauer says that the normal man is two-thirds will and one-third intellect,—in other words, two-thirds made by education and one-third by inheritance. The intellect is often trained so as to enfeeble the will as well as to hinder the development of the physical man. Self-culture may so degenerate into self-indulgence as to destroy individuality and force; and mental health, as a rule, depends upon bodily health and the exercise of self-control." (Folsom.)

5. According to Maudsley, whose views we are developing, *the standards* by which we can measure the perversion are, first, that of the *kind*, which is fixed by the general consent of mankind; and secondly, that of the *individual*, which is estimated by the degree of his previous mental development.

Therefore the individual may frequently perform acts which are not, when compared with his normal standard of action, in any respect whatsoever characteristic of insanity; or the reverse may be the case. There exists, therefore, a standard which the common sense of mankind has established, by means of which all insane perversions are to be compared, measured, and regulated.

By way of illustration, let us glance at certain delusions and the significance thereto attached by such a standard. If a patient tells us that a person came into his room through a key-hole, although he has long been cognizant of the death of such person, would a sane mind experience any difficulty in estimating such a vagary?

So also in other delusions met with during practical observation of the insane,—where they imagine, for instance, that they are made of glass, or as in one case which I recall of a certain lady

who believed that she was an hour-glass, and would request, after the sand had run down during a certain time, "to be turned upside down, in order to allow it to run the other way," in such instances the evidences are so incontrovertible as hardly to need the application of the rule I have just enunciated.

6. *Corroborative proofs* of insanity often exist in cases under examination. Assaults made upon beloved relatives and intimate friends, unaccountable and baseless aversions, especially when accompanied by violent assaults of a homicidal character, which have been perhaps preceded by suicidal propensities, are characteristic of the existence of insanity. Meditative moods, aimless schemes, deep reveries, fits of unusual abstraction, peculiarities and eccentricities of dress, want of tenacity of purpose in persons previously firm, indecent exposures, desire to remain nude, self-mutilation, talking to one's self, squandering property, obscenity, filthy practices, destructive and intemperate habits, are all characteristic of insane people.

7. *The physical symptoms* of insanity should be diligently sought in all equivocal cases of emotional insanity,—viz., headache, insomnia, restlessness, constipation, *et id omne genus*. In the concatenation of circumstances constituting the links of the chain of evidence, the careful, prudent physician will make patient and thorough inquiries in all the directions enumerated before announcing his opinion from the witness-stand. If all heretofore stated be true, the diagnosis of *emotional* insanity is of a very complicated character, and the physician who makes it should divest himself of all partiality and subject the analysis of his case to the closest scrutiny of scientific investigation. In other words, like any other diagnosis in the field of medical inquiry, it should rest solely upon the merits of the naked facts.

DIVISION OF INSANITY INTO TWO PRINCIPAL GROUPS,—(a) AFFECTIVE OR EMOTIONAL INSANITY, (b) IDEATIONAL OR INTELLECTUAL INSANITY. (MAUDSLEY.)

In a former lecture I spoke of the division of insanity into *two primary groups*, naturally characterized by variations of the disease, as affecting either the *words* or the *acts* of an individual. A person may be insane in his *words* or in his *actions*; and this difference admits of a division of insanity into *ideational* and

affective,—ideational insanity being evinced by irrational words, and the affective form by abnormal actions, which are the manifestations or *outcroppings* of insane feelings. These facts we now propose to study; and they are very significant. Indeed, if you have succeeded in realizing this distinction, you have already mastered an important preliminary in the study of this subject.

We have, then, ideational and affective insanity; the latter—also called the *pathetic*—pointing to impulses of perverted volition, the former evinced by irrational conversation or incoherent and erroneous reasoning. We might call this a disease of *ideas* (assuming, however, that they are immaterial), but we have reference, of course, to insane words in consequence of the affection of the cortical cells employed in the elaboration of thought. Hence the individual is ideationally insane, and can no longer think normally, this deficiency being manifested not in his actions but in his words. We find, moreover, that these groupings are not peculiar to any one of the forms of insanity, but to *all* of them, so that we may have either mania or melancholia of the ideational type, or of the affective type, or of *both*. I want you all to understand this fact, which is, according to the best authorities, that in any given case the insanity may partake either of one or of both of these types. Melancholia may therefore exist, and evince only a disturbance of the feelings; but if it coexist with delusions there is then a complication of the ideational with the affective type, and mental action becomes perverted. On the other hand, a person may have mania or melancholia with delusions, and this will necessarily imply an erroneous ideation with aberration of sentiment, as expressed by both words and actions.

I shall not attempt in this lecture to detail the varied and numerous classifications of insanity. Maudsley's division of the subject into two principal groups, the ideational and the emotional, is the greatest advance that has ever been made in this direction, and recommends itself by its intelligibility and simplicity. With the ideational or intellectual variety we have nothing to do in this lecture.

Cases presenting delusions, hallucinations, illusions, dementia, and other perversions of the intellect must necessarily admit of easy interpretation. On the other hand, disturbances of the emotional or affective life may give rise to the greatest discrepancy of

opinion, and, I am sorry to say, even to conflicting and contradictory statements upon the part of medical men. It should be understood that affective, pathetic, and emotional insanity are synonymous expressions defining one primary group, of which *moral* and *impulsive* insanity are only *subdivisions*. *The acts alone* in all their manifestations form the subject of our inquiries.

It is necessary, gentlemen, to impress upon your minds at this juncture a matter somewhat perplexing until properly explained, one that even medical men not sufficiently familiar with the changeful manifestations of insanity but too frequently overlook,—namely, that the existence of intellectual insanity points to a perverted or disturbed condition of *thought*, and wherever this exists a disordered state of ideas will exhibit itself in the *words* of the patient as expressive of morbid *ideas*.

Whenever, on the contrary, the emotional faculties are distorted, discordant *acts* springing from disordered *feelings* force themselves upon our attention as the phenomena to be studied and investigated. How do we manifest our feelings? Is it not by our actions? “Feelings mirror the real nature of the individual, and are the springs of action.” Therefore, as you study perverted *thought* by the expression of incoherent *words*, so are you to study the disturbances which exist in the affective or *emotional* faculties by the development of insane *acts*.

Hence in some forms of insanity all the phenomena must be studied by insane *words*; in other forms—of almost unlimited variety and shading—by insane *actions alone*.

In addition to this it might be stated that the *fundamental state* associated more or less directly with all forms of insanity is *perversion of the affective life*; it is the underlying current which influences and moulds all varieties of the affection.

Persons, at the commencement, generally become insane through a want of proper co-ordination of their feelings. This is the first step outside the limits of mental health. Affective insanity is the deeply-rooted morbid state, of which all the other phenomena are the outcroppings. Hence intellectual insanity is nearly always *preceded* by the *affective* form. Again, if the intellectual form be developed, it is usually in *combination with the affective form*, and, if it be cured, *the affective form generally lingers, and is the last to disappear*. We can now understand that the *affective or emotional*

form of insanity is the fundamental condition with which we must thoroughly familiarize ourselves in order fully to comprehend all the bearings and collateral facts connected with the philosophical study of the general subject. Affective or emotional perversions, therefore, are the precursors of all phenomena culminating in convulsive action and moral irresponsibility. Esquirol holds "moral alienation to be the proper characteristic of mental derangement." "There are madmen," he says, "in whom it is difficult to find any trace of hallucination, but there are none in whom the passions and moral affections are not perverted and destroyed. I have in this particular met with no exceptions." Maudsley, in commenting on this quotation, adds, "This experience is in entire accord with that of every observer of insanity, and with the principles of a sound psychology. It is the feelings that reveal the genuine nature of an individual; it is from their depths that the impulses of action are born, while the intellect guides and controls; and, accordingly, in a perversion of the affective life is revealed a fundamental disorder of the innermost nature, a disorder which will be exhibited in *acts*, as opposed to intellectual disorder, which will be exhibited in *words*."

*"To insist upon the existence of a delusion as a criterion of insanity is to ignore some of the gravest and most dangerous forms of mental disease."**

The importance of an analysis of the *feelings* in the study of insanity cannot, therefore, be exaggerated. The deep, occult subtleties of emotional life constitute the open sesame to psychological inquiry. "By his acts, as well as by his speech, does man utter himself; gesture-language is as natural a mode of expression as speech; and it is in insanity of action that this most dangerous form of affective insanity is expressed,—most dangerous, indeed, because so expressed."

His actions are, therefore, the mirrors of his feelings, the out-comings of the innermost recesses of his nature, of that unconscious activity which so often moulds imperceptibly, but surely, the destinies of man.

Men, therefore, are sometimes unquestionably unable to act rationally, normally, and consistently, being, from deep-seated

* Italics my own.

perversion of the affective life, insane. We all study individuals through their actions. Inborn, unconscious influences "sway the pendulum of life from the cradle to the grave."

We need not wonder that *affective* insanity is the most dangerous form of madness; a man with delusions or other evidence of intellectual aberration is soon locked up, but a man emotionally insane too often commits some overt act of violence before he sufficiently attracts attention to make public opinion demand his sequestration. Not that I attach undue importance to a given act. On the contrary, as I have stated before, *I believe no isolated act in itself capable of proving insanity, no matter how great its enormity, its absence of motive, or its other unaccountable features.*

Suicidal and homicidal propensities, erotomania, kleptomania, pyromania, etc., do not *per se* constitute *distinct* forms of insanity, as was formerly taught. On the contrary, I believe, with Maudsley and Blandford, that such conditions are but purely symptomatic indications of a previously existing insanity.

Blandford says, "Besides the homicidal monomania, we hear of others, as erotomania, kleptomania, and pyromania. Having already stated that I do not consider homicidal monomania to be a specific disease, I still less acknowledge that the acts which these terms indicate proceed from special disorders. They are committed by insane patients of various kinds; the insanity is not likely to be confined to one of these acts, but is sure to be noticeable in other ways, if it exist at all.

"To take the last one mentioned, pyromania, we might as well erect into a special form the window-breaking mania, etc."

Maudsley observes, "So far from the morbid impulse or act constituting insanity, it is but the outward and visible sign or expression of a profound affective derangement, the tendency of which is to manifest itself, not, as ideational insanity does, in words, but in acts, and which for this very reason is much more dangerous than ideational insanity.

"So far from the disease being simply a homicidal or suicidal insanity, it is truly an affective insanity, *one symptom* of which is homicidal or suicidal impulse; the delusion, when there is one, and the homicidal acts are later *symptoms of a deeper-lying disease*; and the morbid manifestation of one may be as little within control as that of the other, or as the suddenly-arising hallucination is.

In the one case he is the victim of a morbid idea ; in the other, of a morbid movement ; in both cases, of a convulsion more or less co-ordinate."

Hence if we attached undue importance to isolated acts we should sometimes mistake for a disease what is only a symptom. To establish a diagnosis under such circumstances necessitates the discovery of other symptoms, otherwise the significance of an isolated act would not be invalidated.

If the public could be made to appreciate this fact, there would be less prejudice against the plea of emotional insanity, and less effort upon the part of lawyers to bring it into contempt by its constant prostitution. The diagnosis of emotional insanity rests necessarily upon so many different proofs as to make it no very easy matter to establish it beyond all possible cavil. Unless it can rest upon the laws of diagnosis which apply to all other diseases, it should not for a moment be regarded as established.

Emotional insanity is therefore known to exist by the history of the case, the existence of hereditary predisposition, the presence of some of the well-known conditions of causation, the change of character, the cessation of social harmony with surroundings, the corroborative circumstances, the impaired judgment of relations, the measuring of the perversion according to an individual standard, or to one accepted by the common sense or the general consent of mankind, the motiveless assaults upon relatives and intimate friends, the existence of some of the physical symptoms of insanity ; in other words, our diagnosis is to be based upon all the above-mentioned states, the etiological conditions, the sequence of symptoms, and the general course of the affection.

If you have grasped these facts, you are in possession, gentlemen, of the most important part of this subject ; and, to recapitulate in a concise manner, I will say that, as in emotional insanity *the feelings are first affected, it is through them we must study all collateral phenomena.* When a truly insane person commits homicide, suicide, rape, or arson, it is the *result* of some serious form of insanity, such acts never constituting the insanity *per se*. They are, therefore, merely the outcroppings or symptomatic manifestations of a morbid condition of the emotional nature ; and delusions are by no means the only evidences distinctive of mental disease, but are simply symptomatic of ideational insanity.

Overt acts of violence indicate a morbid condition of feeling ; incoherent words, a morbid condition of thought ; or, to make it plain, *just as insane words are produced by insane thoughts, so are insane acts produced by insane feelings.*

But some of you may inquire why it is that a person may be the victim of affective insanity only, and not necessarily intellectually insane at the same time.

In reply I would state that such is the case for the same reason that inflammatory conditions sometimes remain *in statu quo*, sometimes advance, sometimes recede. Is inflammation always followed by ulceration, sloughing, suppuration, or gangrene ?

So in a psychological development the fundamental or affective form of insanity may exist alone ; there may never be developed during the case any manifestation of ideational insanity to complicate the situation ; the *disease may not proceed so far.*

DIVISIONS OF AFFECTIVE INSANITY.—MORAL INSANITY.

You have heard, gentlemen, perhaps not very understandingly, that moral insanity and impulsive influences may lead persons instinctively to perform atrocious acts. I will discuss the subjects of moral and impulsive insanity in a few words, first taking up that of moral insanity.

Let me affirm, once for all, that "*moral insanity is never moral depravity,*" and that in order to establish the existence of moral insanity the physician regulates himself by precisely the same rules of diagnosis to which I have so recently referred in the discussion of other forms of insanity. Of late years I have believed, notwithstanding the doctrines of Pritchard, that a careful study of moral insanity will enable us to detect some evidences, although, it must be confessed, often very feeble, of mental weakening. Even the classic cases of Pritchard, who first defined the so-called moral insanity, when carefully examined, will confirm this statement. One thing, however, is certain,—namely, that moral insanity, a revulsion more or less sudden of the emotional nature, of course produced by *disease*, like all other forms of insanity, is preceded by certain physical conditions, such as hereditary taint, epilepsy, suicidal attempts, "the insane temperament," and other influences which are to be taken into consideration. During our tentative

efforts of examination, the diagnosis, therefore, can become established upon an unquestionable basis only by an investigation of all the collateral and combined facts which lead to the development of moral insanity. Blandford says that "we meet with that form of insanity called *moral* when the patient, either on the high road to intellectual insanity or stopping short of delusions, shows his insanity in insane *acts* which he justifies *in general alteration of character and intellectual defect*."*

If a divine distinguished for sanctity, grown gray in the service of the church, suddenly, unaccountably, *without adequate cause*, commenced to steal, became profane and obscene, would we not suspect insanity? On the contrary, innate parsimony, meanness, pusillanimity, suddenly and inexplicably replaced by lavish generosity, princely prodigality, magnanimity, and heroic courage, would more than awaken a suspicion of disease in our minds. Under such circumstances, would not the most sceptical be convinced of the existence of insanity, which always implies a *change in one's normal self in consequence of disease*?

IMPULSIVE INSANITY.

In this condition there is a development of that blind, sudden, instinctive action very analogous to that which sometimes occurs under epileptic influences.

There are strong and irresistible impulses to kill or destroy, or to attempt self-destruction and mutilation. Evidences of mental aberration are generally wanting. The victims of this form of insanity are haunted night and day by such morbid propensities, which they frequently do not fully comprehend or appreciate, and sometimes even make strenuous efforts to resist.

Those outside of asylums will sometimes voluntarily subject themselves to confinement in order to prevent the indulgence of their impulses. Inmates of asylums will in some instances request that mechanical restraint be enforced upon them with the same object in view. The victims of impulsive insanity are relentlessly persecuted by their furious, passionate, and violent propensities. These vicious inclinations constantly tend to express themselves in action, which, sooner or later, completely dethrones

* Italics my own.

the volitional faculty, and manifests itself in explosive violence, or, as Maudsley contends, in convulsive phenomena.

The secret, then, of the psychological interpretation of this subdivision of emotional insanity is *annihilation of the will-power*, which, in point of fact, constitutes the *very essence* of all insane acts. The particular perversions of the affective life are moulded into actions which are not controlled or regulated by the will.

Of course the diagnosis of impulsive insanity is based upon the same irrevocable laws already dwelt upon, and which are the natural basis of all the other varieties of insanity. With every inducement, therefore, to avoid a criminal act, and with a complete knowledge of its wickedness, a man is nevertheless compelled to perform it. "There is no relief for the pent-up destructive energy but in an irresistible utterance of action." It is such cases, therefore, that give rise to serious medico-legal contests.

The impulsive variety, I believe, is never the *first* evidence of insanity. Dr. Blandford says, "I think you will find, if you go to the root of the matter, the act which is supposed to be committed under the influence of insane impulse is rarely, if ever, the first symptom of insanity or brain-affection shown by the alleged lunatic. You may be told by friends that they have never seen any insanity in him; but some people cannot see it in five out of six patients in an asylum. If you get sufficient information, you will probably discover that he has had former attacks, from which he may or may not have been considered as recovered." Sankey, as quoted by Sheppard, says, "I have taken the precaution to read a large collection of reports, published from time to time in the *Annales Médico-Psychologiques*, upon the state of the mind of persons accused of different acts of violence. *I have never yet discovered a case in which an act of violence was committed by a lunatic as his first insane act.*"* There are cases in which the patient was not considered insane by his own relatives, or by inexperienced practitioners, but who, on the closer scrutiny of the physician who had studied insanity, was clearly proven to have been so for a long period." The burden of proof as to the existence of impulsive insanity, therefore, *rests upon the ordinary laws of medical diagnosis*. Maudsley says that "there are not only

* Italics my own.

perverted appetites, but there are perverted feelings and desires, rendering the individual a complete discord in the social organization. The morbid appetites and feelings of hysterical women and the singular longings of pregnancy are mild examples of a perversion of the manner of feeling and desire, which may reach the outrageous form of morbid appetite."

As an illustration of impulsive insanity I quote the following from Maudsley: "An old lady, aged seventy-two, who had several members of her family insane, was afflicted with recurring paroxysms of convulsive excitement, in which she always made desperate attempts to strangle her daughter, who was very attentive to her, and of whom she was very fond. Usually she sat quiet, depressed and moaning because of her condition, and was apparently so feeble as scarcely to be able to move. Suddenly she would start up in great excitement, and, shrieking out that she must do it, make a rush upon her daughter, that she might strangle her. During the paroxysm she was so strong and writhed so actively that one person could not hold her; but after a few minutes she sank down exhausted, and, panting for breath, would exclaim, 'There! there! I told you; you would not believe how bad I was.' No one could detect any delusion in her mind.

"The paroxysm had all the appearance of a mental convulsion, and, had she unhappily succeeded in her frantic attempts, it would certainly have been impossible to say honestly that she did not know that it was wrong to strangle her daughter. In fact, it was because of her horrible propensity to so wrong an act that she was so wretched.

"It is a sufficiently striking commentary on the present state of the English law, that, had this patient succeeded in taking her daughter's life, sentence of execution must have been passed, and might have been carried into effect, notwithstanding she was so entirely insane and irresponsible."

In the family of Baron von Humboldt, on one occasion his wife, on returning home, found the servant in tears, who immediately threw herself on her knees and confessed her inability to remain in the house on account of an irresistible desire to kill their little child. This propensity she attributed to the whiteness of its flesh, which incited her homicidal tendency whenever, whilst bathing and dressing the child, she was left alone with it.

Maudsley quotes Schenck as relating "the history of a pregnant female, in whom the sight of a bare arm of a baker excited so great a desire to bite and devour it that she compelled her husband to offer money to the baker to allow her only a bite or two from his arm. He mentions another pregnant female, who had such an urgent desire to eat the flesh of her husband that she killed him and *pickled* the flesh, that it might serve for *several* banquets."*

I would surpass the limit allotted to this lecture were I to furnish further illustrations of impulsive insanity. You can consult the literature of the subject, which abounds in them.

MEDICO-LEGAL ASPECTS OF EMOTIONAL INSANITY.

To the community at large, the plea of insanity is one of the utmost importance. The medical witness, therefore, has a *double* duty to perform,—justice to the community, and justice to the prisoner at the bar: society demands protection against crime and violence, while the prisoner, if *insane*, has not forfeited his claims upon our humanity. The abuse of the plea of insanity at the present day is fraught with great evils, and the plea itself is therefore constantly derided by the press and the public.

Yet I am convinced that you will not dispute the fact that where real insanity is *proved* it would be most unjust to subject to the extreme measures of the law a person whose volitional powers are morbidly impaired or destroyed, and who therefore must be morally irresponsible.

Hence I think, with Forbes Winslow, in his "Plea of Insanity," that it would be proper and just to leave the adjudication of such cases to a commission of medical experts. This was done with very satisfactory results by the late Judge Primm, of this city, in the Cronenbold case, thereby saving the State the expense of a long trial and doubtful conviction. He organized a commission of five experts, who were empowered to examine witnesses under oath, and upon the return they made Cronenbold was sent to an insane asylum, where he still remains.

I am firmly convinced that simple justice requires, whenever a man, after committing a homicide, is liberated upon a plea of insanity, that *ipso facto* he should be sent to a lunatic asylum for

* Italics my own.

life, because in case of supposed recovery no one could assume the responsibility of affirming that sooner or later his dangerous disease would not recur. On the contrary, every physician is aware that one attack of insanity predisposes to subsequent attacks, and that therefore, from the very nature of the act, and the probability of its recurrence from the very nature of this disease, if a man have once deliberately taken the life of a fellow-creature in consequence of want of proper volitional control due to the disease of the brain we term insanity, then for and during his life he should be deprived of liberty and never again be afforded a chance to perpetrate similar deeds.

The liberation of Joseph Fore in this city, after being acquitted upon a plea of emotional insanity, was an outrage and injustice which no terms are strong enough to condemn. His tragic death and subsequent events also conclusively proved the dangerous and destructive tendencies of this man, whose days should have been passed among the insane. Physicians therefore should pass upon the plea of insanity, not lawyers or judges; the former study the phenomena of insanity with patience and accuracy, while the latter are utterly ignorant of the necessary fundamental knowledge.

Physicians are constantly with the insane; they examine the brain-action of those afflicted with mental disease, after having made themselves thoroughly acquainted with the laws of brain physiology. Clinical, physiological, and pathological research peculiarly constitutes them the interpreters of the manifestations and phenomena of the mind diseased. Would it not be absurd to require a physician to decide some abstruse and important legal question, some mechanical problem, or metaphysical proposition?

Who is more familiar with all the phases and complications of mental alienation than the psychological physician? Therefore, is it to be wondered at that such irreconcilable antagonisms exist between medical and legal minds, and such vexatious contentions upon this sea of emotional insanity? These antagonisms are the more to be regretted because those who from their studies and vocation must necessarily keep abreast of the progress constantly being made in the field of psychological medicine, who are particularly and practically adapted, therefore, to examine into these questions, continually find themselves opposed and thwarted in their aims by minds more guided by legal acumen

than enlightened by the scientific status of the questions involved. Hence the breach is always widening. The non-professional observer of the contest from day to day grows more sceptical, humanity suffers, and the discoveries of science are rendered less available.

We have already referred to the question of *motive*, but in this particular connection a few more remarks may be added, in view of the importance of this subject.

When a son kills his father, not influenced by passion, the desire of inheritance, or any other discoverable cause, a strong suspicion and presumption of insanity will exist. When a mother destroys her children to send them to heaven, the same doubt of her sanity will suggest itself. But, gentlemen, you must be made aware that the insane are influenced by the ordinary motives of human action equally with persons of incontestable sanity.

If you visit an asylum and strike one of the patients, he will resist your violence and strike back; if your bearing be insulting, it will immediately provoke resentment. In the ordinary management and discipline of institutions for the insane these facts are recognized, and the entire moral treatment is thereby influenced. They are therefore punished or rewarded in a paternal manner when the necessities of their case demand such a recourse, otherwise the rules and moral discipline which form the most important factors in promoting their restoration would be without success. Hence the maxim never to be lost sight of in dealing with the insane consists in "*kindness and firmness*," inasmuch as they allow themselves to be swayed by motives like other people.

Then, again, there are sane people the interpretation of whose motives is but too often an inexplicable mystery, and we should have to plunge very deep into the labyrinths of the human heart, into the innermost recesses of the soul, to be always successful in our efforts in this direction. "It is assumed or implied," says Dr. Taylor, with great justice, "that sane men never commit a crime without an apparent motive, or one of delusive nature only in the perpetration of a criminal act. If these positions were true, it would be very easy to distinguish a sane from an insane criminal; but the rule wholly fails in practice. In the first place, *non-discovery* is here taken as a proof of the *non-existence* of a motive; while it is undoubted that motives may exist for many atrocious

criminal acts without our being able to discover them, a fact proved by the numerous recorded confessions of criminals before execution, in cases of which, until these confessions were made, no motive for the perpetration of the crime had appeared to the acutest minds."*

Should lunatics be hanged? It seems hardly necessary in this enlightened age to discuss such a question, yet the subject is occasionally re-agitated, notwithstanding its senseless barbarity. Some hold that, as lunatics are dangerous, expensive, and of no possible benefit to the community, *pro bono publico* they should be hanged as soon as they perpetrate a crime. The atrocious inhumanity of such a sentiment is as disgraceful as it is brutal.

Incarceration of the insane in asylums cannot ward off the same fate from others. Example can exert no influence in the prevention of disease. Forbes Winslow, commenting on this subject, maintains that we might as well insist upon inflicting capital punishment upon epileptics because of their misfortune. But would this prevent the development of epilepsy in a single individual?

As regards the *plea of insanity* Lord Hale's views were extreme. He held that "there must be defect of the understanding, unequivocal and plain."

Lord Coke held "that whoever by sickness, grief, or other accident wholly loses *his memory and understanding*" is *non compos mentis*.

Lord Chief Justice Mansfield, in the trial of Bellingham, charged that "the single question was whether, at the time this act was committed, he possessed a sufficient degree of understanding to distinguish good from evil, *right from wrong*, and whether murder was a crime not only against the laws of God, but the law of his country."

Lord Erskine considers "*delusion* where there is no frenzy" to be the true character of insanity. "Viewed, however, as a principle of law, the delusion and act should be connected." *Delusion* we admit to be strong presumptive proof of insanity, and is insanity in almost ninety-nine cases out of one hundred. "*Delusion* exceptionally," says Winslow, "may be present and not be incompatible with sanity." If we take *delusion* as a criterion, we are liable to

* Wharton and Stillé's Medical Jurisprudence.

ignore some very dangerous forms of insanity, because there are many serious manifestations of the insane temperament, as well as many affective forms of insanity, which will attract our attention only through actions following the irresistible impulse of distorted feelings. Delusions, therefore, cannot be correctly considered a criterion of insanity, since it may indubitably exist without their presence. This is a very important fact, with which you should be acquainted, because for a long time in England, as well as in America, delusion was always considered a *sine qua non* of insanity; and even to-day many prejudiced lawyers consider that its absence entirely invalidates the plea of insanity, and many very important judicial decisions, involving property, and even life, were erroneously based upon this principle. To-day the old doctrine is rejected, almost entirely, by the best authorities, and the new one is receiving no little legal sanction; and if you adhere to obsolete ideas you will necessarily aid in impeding psychological progress.

According to the 64th article of the French penal code, "Il n'y a ni crime ni délit lorsque le prévenu était en état de démence au temps de l'action."

Forbes Winslow, in commenting upon the above opinions, states, "I am disposed to express my complete concurrence in the views of Dr. Haslam on this point, that 'it is not the province of medical writers to pronounce an opinion as to the prisoner's capability of distinguishing right from wrong. It is the duty of the medical man, when called upon to give evidence in the court of law, to state whether he considers insanity to be present in any given case, not to ascertain the *quantity of reason** which the person imputed to be insane may or may not possess.'"

"If it should be presumed," says Dr. Haslam,† "that any medical practitioner is able to penetrate into the recesses of a lunatic's mind at the moment he committed the outrage, to view the internal play of obtruding thoughts and contending motives, and to depose that he knew the good and evil, right and wrong, he was about to commit, it must be confessed that his knowledge is beyond the circuit of our attainment. It is sufficient for the medical practitioner to know that such a person's mind is deranged, and that

* Italics my own.

† Forbes Winslow, "Plea of Insanity in Criminal Cases."

such a state of insanity will be sufficient to account for the irregularity of his actions, and that in a sound mind the same conduct would be deemed criminal. If violence be inflicted by such a person during a paroxysm of rage, there is no acuteness of metaphysical investigation which can trace the succession of thoughts and the impulses by which he is goaded to the accomplishment of his purpose."

I am sure, gentlemen, that you will perceive from what has been insisted upon in the first part of this lecture that the most dangerous forms of insanity are those in which neither illusions, delusions, hallucinations, nor perversions of the intellect generally, are discoverable.

As regards the recognition of the difference between *right and wrong*, it is now an indisputable fact that nearly all the insane make the distinction without difficulty.

I here cite from Folsom on "Mental Diseases" the following opinions:

"Lord Bramwell once said that 'insanity is strong but not conclusive evidence of innocence;' and Lord Blackburn has stated that 'the jury must decide in each individual case whether the *disease of the mind* or the *criminal will* was the cause of the crime.' The position of Sir James Stephen, in his 'History of the Criminal Laws in England,' best states the most recent views of irresponsibility,—namely, that 'no act is a crime if the person who does it is, at the time when it is done, prevented either by defective mental power or by any disease affecting his mind from controlling his own conduct, unless the loss of the power of control has been produced by his own default.' He says that a man laboring under such a defect of reason that he does not know that he is doing what is wrong may be defined as one deprived, by disease affecting the mind, of the power of passing a rational judgment on the moral character of the act which he meant to do. There are persons too insane to make a valid will by virtue of a single delusion, whose right to vote, under the law prohibiting the insane from voting, would not be questioned. Another might not be held responsible for crime, and still make a contract involving the rights of others besides himself that would hold in law."

"Bucknill's recent medico-legal definition of insanity is, incapacitating weakness or derangement of mind produced by disease;

meaning, in criminal cases, inability of abstaining from the criminal act, which would be expressed by Lord Bramwell's test, 'Could he help it?' Bucknill suggests as an amendment to the law of England that no act is a crime if the person who does it is at the time incapable of not doing it by reason of idiocy or of disease affecting the mind." (Folsom.)

The volitional centres are invariably affected in all forms of insanity. *Hence in every judicial charge the question to be determined is as to whether or not the criminal pleading insanity, while recognizing the difference between right and wrong, is able or unable to control his action.*

Baillarger states that the essential element of insanity is *loss of free will*.

Ball, of Paris, describes an insane man as "one who in consequence of a profound disturbance of the mental faculties has lost more or less completely his free will and has ceased thereby to be responsible for his actions."

It would be as rational to punish a school-boy whose antics and grimaces, the result of chorea, are a source of laughter and distraction to his school-mates, as to inflict punishment upon the insane criminal, who, knowing the difference between right and wrong, has it not in his power to execute that which his judgment dictates. One is under the dominant influence of "insanity of the muscles," the other is under the influence of *insanity of the will*.^{*} To punish one would be as cruel as to punish the other.

One of the most powerful forces which can affect the workings of the intellect is that derived from the presence and influence of insanity, which weakens and prevents and even in many cases annihilates the faculties of volition. Without the exercise of the regulating power of volition, mental convulsion and discordant, incoordinated, and automatic actions are inevitable with blind, instinctive, and irresponsible results. No moral responsibility, therefore, can exist where insanity subverts the directing and controlling power of the will. It gives me pleasure to state that the Supreme Court of the United States in a recent decision maintains that the *presence or absence of will-power*, in the plea of insanity, is the question upon

^{*} *Faber Winslow.*

which the existence of criminality and responsibility exclusively depends.

Insanity as an extenuating plea in cases of murder requires that the previous condition of the individual's life should be investigated. Winslow declares "it necessary to inquire whether the person has at any previous period of his life manifested any signs of mental derangement; if such be the fact, it ought to constitute a *prima facie* case in his favor."

Isolated facts, when introduced in support of the plea, with no exceptions, in my opinion, notwithstanding the weight of the distinguished authority just quoted, should be valueless, unless the insanity can be established by a *concurrence* of indisputable and conclusive facts resting upon the broad principles of medical diagnosis. Upon this point I cannot too strongly insist. In order to facilitate the interpretation of cases in which the plea of emotional insanity is made, Dr. Pritchard lays down the following rules, based upon the conjoint observations of Esquirol and himself:

1. "Acts of homicide perpetrated or attempted by insane persons have generally been preceded by other striking peculiarities of action, noted in the conduct of these individuals, often by a total change of character.

2. "The same individuals have been discovered in many instances to have attempted suicide, to have expressed a wish for death; sometimes they have begged to be executed as criminals.

3. "These acts are without motive, they are in opposition to the known influences of all human motives. A man murders his wife and children, though known to have been tenderly attached to them; a mother destroys her infant.

4. "The subsequent conduct of the unfortunate individual is generally characteristic of his state. He seeks no escape or flight, delivers himself up to justice, acknowledges the crime laid to his charge, describes the state of mind which led to its perpetration; or he remains stupefied and overcome by a horrible consciousness of having been the agent in an atrocious deed.

5. "The murderer generally has accomplices in vice and crime; there are assignable inducements which led to the commission, motives of self-interest, of revenge, displaying wickedness premeditated. In some instances the acts of the madman are pre-

meditated, but his premeditation is peculiar and characteristic. There is also a presumption of insanity, where the individual has either been previously insane, or affected by epilepsy."*

It is therefore self-evident, gentlemen, from all that has been said, that while insanity in general is "*a reasoning unreason*," yet in the emotional or affective form of that disease we must ignore the particular morbid manifestations of thought as expressed in words, and confine ourselves to the study of the actions which portray a disordered condition of feeling. The plea of emotional insanity, we have ascertained, far from being one of a trivial nature and easily maintained, as popular prejudice might induce you to believe, is of an intricate and complicated character, requiring for its establishment a thorough knowledge of the philosophy of the general subject of insanity and no ordinary powers of observation and analysis. In addition to this, the physician investigating the phenomena of emotional insanity should possess earnestness, perfect candor, and an *unbiassed* mind. Leaning in one direction or another redounds to the discredit of science and the still further degradation of a most legitimate plea, which, if resting upon the basis of a careful diagnosis, is sound in theory and practice and of great service in a philanthropic point of view. When abused, as it is but too often, the plea can result only in the perversion of justice and in professional disgrace, and in detrimental effects reaching not only the individual but also society at large.

* Forbes Winslow, "Plea of Insanity in Criminal Cases."

LECTURE XV.

INSANITY—continued.—MELANCHOLIA.

Phenomena—"Concrete Form of Misery"—Difference between Melancholia and Mania—Lypemania and Pantophobia—"Furor Melancholicus"—Hypochondriacal Melancholia, Phenomena of; Case of—Another Form of Melancholia—Distention and Torpor of Colon—Tendency to Suicide—Melancholia Attonita—*Folie Circulaire*, or "Circular Insanity"—Paroxysmal Violence not necessarily an Evidence of Insanity—Affective Form rarely absent—Moral Treatment: Evil Effects of Delay, Beneficial Effects of Asylum Treatment—Medical Treatment: Opium, Aloetic Laxatives, Tonics, Alcoholic Stimulants, Sulfonal for Insomnia, Rhamnus Frangula, Phenacetin and Citrate of Caffeine in Posterior Cervical Pains of Melancholia, Repression of Menstruation.

GENTLEMEN,—To-night we will commence the consideration of the other forms of insanity. The first one I shall speak of, and one with which you will frequently meet, is melancholia. Melancholia, as the name denotes, is characterized by great mental depression, excessive grief, and painful sadness, and owes its origin "to the usual *dual cause*," as M. Allen Starr properly observes, —viz., "*physical ill health and mental strain*." It belongs, for the most part, to the *affective* or *pathetic* variety of insanity, as it relates more to the feelings of the individual than to his intellect.

The first phenomena to be observed are, peculiar mental depression, vague and indefinable oppression, strange and indescribable sorrow. There is a state of gloom which the patient cannot shake off, "an oppression of the self," and his despondency is to himself something altogether unaccountable; he is at a loss to explain why he is so sad, or why he experiences this vague and indefinite condition; in short, he is entirely overwhelmed and completely overburdened, life becomes a misery, and existence is unendurable. This general *affective* dejection may constitute the entire morbid state, no delusions existing, the *feelings* only being perverted without any implication of the intellect. Delusions produce a form of insanity in which we always presuppose an affection of the intellect; that is ideational insanity. As

Maudsley says, after the mental depression of melancholia has existed for some time, "*a concrete form of misery*" is produced; the patient seems to assume some fixed and settled idea, or develops a terrible, all-absorbing delusion. This *concreteness* is indicated by delusion, and suggesting, as it does, a hypothetical cause for the overpowering anxiety of the mind, far from increasing the grief or depression, seems generally to afford relief. This delusion, however, bears no adequate relation to the intensity of the melancholia. In some persons it appears as a very trivial matter, in others it is "expressive of some great fear or suffering." We find some melancholic patients laboring under the belief that they have committed some grave offence, or that they are guilty of "the unpardonable sin." They imagine that they are suffering the torments of the damned, and experience these tortures for some trivial delinquency, which ideas are absurd, of course, but greatly exaggerate their distress. In melancholia, which strictly pertains to the affective variety of insanity, the morbid manifestations are mainly evinced in the actions of the patient, while in mania, the intellect being affected, the disturbances are chiefly exhibited in the words, as expressive of thoughts.

Melancholia, as met in general practice, at the bedside, or in the hospitals, is capable of being separated into two original groups,—*lypemia* and *pantophobia*. *Lypemia* consists of melancholia that is always attended by delusions. *Pantophobia* refers to a condition of the patient which is characterized by a constant dread and fear of everything, without any definite cause for apprehension. The latter is melancholia without "concrete form." Remember that this division is made by Maudsley simply for practical purposes. I have already told you that there are two grand primary divisions of insanity,—the *affective* form, relating to morbid feelings, and the *ideational*, relating to perverted intellection. *All cerebral disorders resulting in insanity come under one or the other, or under both, of these forms.* This being the case, it follows that just as we have melancholia without intellectual insanity, we sometimes have mania with disordered actions only, and of purely *affective* nature.

When mania exists, it is almost invariably in the form of intellectual insanity; still, the precursory manifestations are emotional, and we have acts expressive of deranged sentiments, affections,

habits, etc., prior to other developments. In mania, therefore, when affective in character, the perverted feelings are preternaturally excited,—in contradistinction to melancholia, in which they are depressed. Therefore, when the insane feelings are *excited*, there is an affective *mania*; when they are depressed, there is an affective *melancholia*. If now in affective melancholia delusions supervene, the intellectual powers becoming impaired, the faculty of reasoning is involved, the conversation is more or less irrational, and then intellectual insanity has appeared and complicated the case.

The first phenomena of melancholia refer to disturbed feelings, through grief, sadness, despondency, etc., without any intellectual derangements. Of course the patient is not, as yet, ideationally insane; but delusions may sooner or later complicate the case, and we then have ideational insanity, and the patient's abnormal condition will be manifested alike in words and in actions.

Another subdivision of melancholia is the *hypochondriacal*. In this form we find the individual studying, criticising, analyzing, examining, and investigating his own real or supposed bodily ailments. He imagines himself the victim of some disease; perhaps he has a "fluttering of the heart, a film before his eyes," etc., and all sorts of indescribable troubles annoy him. He watches these hypothetical symptoms and interprets them in his own way, and, when consulting a physician, will often give explanations of his disease which are really remarkable. It occurs in many instances that a melancholic patient may be only slightly hypochondriacal, and yet be unable to attend to his business or pursue the ordinary vocations of life. Of course this constant, self-absorbing pre-occupation becomes a source of disease, and results disastrously, by inducing certain changes in one or more organs. The incessant concentration of the mind on the feelings develops delusions, and, acting under the influence of such perverted ideas, the patient may be led to the commission of rash actions, with terrible consequences. Whence do our actions spring? Whence our impulses? They originate from the feelings; hence the perverted feelings, stimulated by a morbid influence, will sometimes induce a patient to commit suddenly some terrible crime, and this may occasionally happen even in some form of hypochondriasis. It has occurred, as related by Maudsley, that a patient cut open his abdomen with

a piece of glass, penetrating the intestine, simply "to let out the gas."

Now, when the feelings are thus perverted so as to constitute disease, whether slight or intense, it gives the physician great annoyance; and many hypochondriacs out of asylums will come and torture you by a detailed account of their troubles. The face will be pinched and anxious, the story related you will perhaps have heard a hundred times and know by heart, and yet you must patiently listen. To treat such people is, indeed, a task, though it is at times exceedingly interesting to observe their peculiarities. A short time ago I had under treatment a hypochondriacal gentleman, who stated that he was suffering from muscular rheumatism to such an extent that all locomotion was impeded. This pain had resisted iodide of potassium, salicylate of sodium, and all other treatment. The other evening I saw him; he recollected that he wanted to go to a certain place of amusement that very night, which inclination so absorbed his thoughts that he overcame his morbid feelings, jumped up suddenly, walked off briskly, and was cured, until, his interest being no longer excited by extraneous matters, the old trouble returned, because his thoughts reverted to the same morbid channels. All cases of insanity are not sent to the asylum for treatment. Some wealthy families will often seriously object to, and strenuously oppose, a removal of one of their members, their station in life enabling them to furnish all possible conveniences and comforts to the patient at home.

There is another form of melancholia, which you may meet with in the asylums as well as in private practice, and which you should be able to recognize. I refer to *melancholia with excitement*, or "*furor melancholicus*," which is sometimes not readily distinguished from mania. Delusions will appear, and the patient become very prone to violence; a dangerous homicidal propensity will be developed, and intense mental anguish may culminate in suicide. This morbid tendency to the consummation of some terrible act is engendered by the desire to relieve the crushing depression. I remember a case in an asylum, where such a patient, who had previously been considered harmless, actually knocked a man down and beat his brains out with a chair before assistance could be obtained. When asked by the coroner his reason for committing this crime, and if he had any spite against his victim,

or had any previous quarrel with him, he simply answered, no ; that, on the contrary, they had always been good friends, and it was only the pent-up feeling, the excruciating mental torture, which he by *some act desired to relieve*, that caused him to commit this deed of blood. Such being the possible consequences of perverted emotions, you will readily understand why this affective form of insanity is so much to be dreaded.

Another danger in melancholia, and one against which you must always be upon your guard, is a tendency to *suicide*. This impulse has to be thwarted ; and the actions of all melancholic patients are to be closely watched on this account, as the intensity of their mental suffering is sometimes so great that they frequently attempt, and but too often succeed in accomplishing, their own destruction. They resort to cunning artifices of every description for the accomplishment of their purpose, picking up pins and needles from the floor, and concealing all sorts of objects, with which at night they will attempt to destroy themselves. I have known them to secrete a table-knife, to use at the first opportunity when unobserved ; and in my own experience I recollect a patient who cut his throat from ear to ear with a knife which he had slyly abstracted from the dinner-table and sharpened on the window-sill. Indeed, the more closely they are watched, especially if they suspect it, the more persistent are they in their efforts at concealment and deception. Hence a *sleepless vigilance* alone will be successful in frustrating their destructive propensities. It was formerly taught that suicide was one of the dreaded terminations of melancholia, but it is now generally admitted that it is a *symptom* of the disease, and that the person should never be left one minute alone.

“So far from the morbid impulse or act constituting the insanity, it is but the outward and visible sign or expression of a profound affective derangement, the tendency of which is to manifest itself, not, like ideational insanity, in *words*, but in *acts*, and which for this very reason is much more dangerous than ideational insanity. So far from the disease being simply a homicidal or suicidal insanity, it is truly an affective insanity, one symptom of which is homicidal or suicidal impulse : the delusion, when there is one, and the homicidal act are both symptoms of the disease ; and the morbid manifestation of one may be as little under control as of

the other, or as the suddenly arising hallucination. In the one case the patient is the victim of a morbid idea ; in the other, of a morbid movement ; in both cases, of a convulsion more or less co-ordinated. Where the disease is less acute, it is the feeling of this affective perversion that sometimes drives the melancholic to commit murder in order to be hanged, or impels a mother to murder her children in order to send them from misery on earth to happiness in heaven. It admits of no question whatsoever, and should therefore be borne clearly in mind, that the calmest melancholic is liable to periodical unaccountable exacerbations of disease, during the paroxysms of which he may perpetrate violence against himself or others ; a wonderful relief, and even an apparent sanity, with endeavor to escape penal consequences, sometimes following the accomplishment of the act." (Maudsley.)

Another form of melancholia, and one which might be confounded with dementia, is *melancholia attonita*, or melancholia with stupor. Life in such patients seems to be purely vegetative ; they will take no food, refusing even to swallow it when it is placed in their mouths. At times they will resist all efforts to introduce aliment into their stomachs, from fear of being poisoned, imagining that some relative or friend wishes to kill them. The peculiarity of this variety is that the patient is in a seemingly cataleptic condition,—a *quasi* stupor or somnolent inertia,—and on account of his immobility forcibly reminds you of a statue. Once having assumed a position, the patient will maintain it for hours, perhaps for a whole day, or until utterly exhausted. There is a striking inanimation, the organic functions appear almost to be held in abeyance, the animal actions are suspended, and the face is a blank, without the slightest expression or play of emotion. There is no consciousness of time, locality, or personal identity ; the patient has not the least idea of his surroundings, sometimes involuntarily swallowing anything placed upon his tongue, and at other times it will be impossible to make him eat, until you at last resort to the stomach-pump, introducing food mechanically. At times the patient will temporarily revive, like a person awakening from a sound and protracted slumber, during which he has apparently been dreaming, and will make inquiries which undoubtedly point to previous unconsciousness. His mind has been engrossed in one terrible delusion, which wholly absorbed his

faculties ; but one feeling, one idea, has prevailed, in which his whole being has been concentrated, and since, as Dr. Maudsley remarks, to be wrapped up in one sensation would be equivalent to the possession of none, we can understand the profundity of the patient's lethargy. This form of melancholia is really painful to witness, and the immobile and cadaverous appearance of its victims is well calculated to arouse sympathy.

Still another form of insanity to be considered is what is termed by the French *folie circulaire*, or "*circular insanity*." In this form there is a certain alternation in the manifestations from melancholia to mania, and it may be a difficult matter to differentiate between the two diseases. At one time there may be an exacerbation characteristic of mania, followed by a period of mental depression. I have seen this type of insanity develop itself towards the termination of protracted cases of melancholia, and in a case of five years' duration of the latter affection, now, strange to relate, convalescent, *folie circulaire* was the immediate precursor of the present favorable condition. The case to which I refer was that of a distinguished divine, and was the most profound I have ever witnessed, being for eighteen months, in the earlier periods, *melancholia attonita*. *Folie circulaire*, however, very rarely follows this satisfactory course, as in many cases of inveterate and incurable insanity it will be present.

Perhaps it would be well for me to caution you against supposing that acts of paroxysmal violence necessarily constitute a feature of affective insanity. This would be an erroneous conclusion, as such explosions are purely symptomatic, really forming but a single feature of the malady, and in some cases are entirely absent : just as in ideational insanity we do not necessarily find delusions, though they are generally present, so in some cases of affective disorder murderous and suicidal and other morbid propensities may not be manifested. The old subdivision of certain forms of insanity into pyromania, erotomania, suicidal and homicidal mania, etc., was quite unfortunate, and calculated to lead to erroneous conclusions : we now know that the propensities constituting those states are only *symptoms* or outcroppings common to many, and hence not in the least peculiar to any special form of insanity. As I have said in previous lectures, the most dangerous forms of insanity may and often do exist without delusion ;

so also these last-mentioned conditions are often entirely absent. Madmen are frequently able to *regulate their conversation* perfectly, speak coherently, and even astonish you with the brilliancy of their thoughts, but they cannot *control their actions*; hence the affective form is rarely absent, but generally pervades all the forms and varieties of insanity.

To repeat: in insanity the affective type is almost always at the bottom of the difficulty, developing perverted emotions and change of habits. This state may be very dangerous without any evidence of intellectual disorder. In short, it usually complicates all varieties of insanity, preceding, accompanying, or following them, thereby greatly influencing their course and profoundly modifying their manifestations. These are very important facts, and after you understand them a great part of the subject of insanity will become clearer to your minds.

It is well to remember that the moral treatment is the most essential of all the influences requisite in the management of this form of insanity. The moral treatment is indispensable, because perversion of sentiment is the fundamental condition of the disease. In what does this treatment consist? In isolation, seclusion, *firmness* combined with *kindness*, and discipline of mind and body. I have already told you that when a person is insane he is no longer in harmony with his social relations; there is something out of gear, and he may become a source of danger to himself and to others. He should, therefore, be isolated, and treated by those who understand his affection. Metaphorically speaking, the loose screw should be accurately adjusted, and the machinery of the brain be oiled and reconstructed, that future harmony may prevail. As general practitioners it is your first duty to know this, and I cannot too persistently dwell upon it. Many an unfortunate being, through neglect, injustice, or ignorance, has been doomed to the miseries of chronic insanity, compared to which all other sufferings are small, and the agony of death itself infinitely preferable. Yes, death is a thousand times more desirable than a life embittered by chronic insanity and rendered desperate and horrible by the perpetuity of its wretchedness. If this practical knowledge were the only benefit you have derived from these lectures, you would have gained much, and would have acquired information which, though apparently of little value, is

very important in its bearings from a humane point of view. When the patient, consequently, is in the inceptive stages and the disease is still acute, he should be immediately treated or sent to an asylum, in order to prevent the occurrence of hopeless mischief. Why is it that the number of inmates of county and State asylums is constantly swelling? Is it not due to the neglect in this regard of physicians, or to unfortunate legislation and the endless amount of "red tape," by which the destitute are left without proper treatment until the disease is no longer acute, but chronic,—its intractability being in exact relation to its duration? By this time the patient has become not only a permanent burden to his family but also a life-long curse to himself, and the asylum has finally to open its doors for him to enter, never again, perhaps, for him to go out. Begin your treatment early, therefore, remembering that when three months have passed insanity ceases to be acute, and that success depends upon immediate and energetic treatment. I may here state my thorough conviction that some cases of melancholia may recover without being sent to an asylum; but this is exceptional, and does not invalidate the rule. Patients may sometimes be effectively treated at their homes, but not usually; for, while a rich man may command a comfortable residence and faithful attendants, and may secure more eminent physicians to treat him, how many can afford these expensive conditions of home treatment? and is not the number of the suffering and poor infinitely greater? Moreover, even under the best home treatment, from the inexperience of nurses, suicide will but too often result.

There is often a prejudice against sending persons to lunatic asylums, from the fear that their sudden removal to such associations may prove pernicious. On the contrary, the moral effect is often excellent: the patient recoils; there is a beneficial shock, which occasions him to enter into himself and reflect, "Why am I here, in the company of persons so plainly mad?" This causes an introspection, with a most salutary effect, to which the order and discipline of the place are distinctly auxiliary. The moral measures also concur in teaching the patient to exercise a certain amount of self-control and to make efforts towards recovery. You may exhaust the therapeutic resources of the pharmacopœia, and, if the patient do not try to get well, he will not improve;

and it is this desire of restoration for which I look as the first auspicious sign of dawning reason. If the patient evince no energy, he will not get well ; but if he try to disperse the mental mist which surrounds him, he will frequently progress favorably, and his endeavors will to a great extent be advanced by the moral treatment he receives.

The subjacent *pathological* condition, I fully believe, as has long been taught, is *cerebral anæmia*.

TREATMENT.

The insane should be treated like children, with kindness and firmness, with uniform affection and courtesy ; but the necessary exactions must be unswervingly enforced, and, like children, they should be rewarded for their good behavior and rebuked for their disobedience. They often will do better, or at least, like children, will try to do better. When depressed, you should encourage them and relieve their mental anxiety, and when unruly, you must check them, but without undue severity. This is far more important than the *medical treatment*, in regard to which I must say that I have not unbounded faith. One thing is certain, as maintained by Schroeder van der Kolk, *that melancholia is often due to a loaded colon, accompanied by obstinate constipation*. In such cases an aloetic laxative, or Rhamnus frangula, the laxative *par excellence*, will generally relieve the constipation and cause mental cheerfulness to reappear. Since the publication of the last edition of these lectures, in which I condemned the use of opium, I have returned with more confidence to this *classic* treatment of melancholia. Judiciously handled, it produces more permanent and beneficial effects than any other remedy with which we are acquainted.

As in the affective variety of insanity there are great mental sorrow and physical prostration, alcoholic stimulants can be beneficially administered. Melancholic patients bear them remarkably well, and to an extent truly astonishing. According to Blandford, such patients should receive alcoholic stimulants at every meal,—either rum, brandy, or whiskey. Of course there is a medium in everything, and so in the administration of these stimulants you should bear in mind that what may be too little for one may be too much for another. I have always found the breath the best guide, in this as well as in acute and febrile diseases, in determining

the proper regulation of the remedy: if surcharged with alcoholic fumes, all the alcohol is evidently not appropriated, the system is surfeited, and its supply should be correspondingly curtailed. Headache of the incipient stage, and the "posterior cervical pain" described by Landon C. Gray, may be relieved by phenacetin. Headaches and neuralgias dependent upon cerebral anæmia are rapidly and magically alleviated by the following:

R Phenacetin., gr. x;
Caffein. citrat., gr. iii.
M. et ft. chart. no. i.
S.—Take in one dose.

Conquer the insomnia at night by the judicious administration of sulfonal or paraldehyde, remove as far as you can all causes of the disease, give tonics, and, by a liberal administration of milk and nitrogenized food in a liquid and concentrated form, seek to maintain the forces in the best possible condition to resist the inroads of the malady. It should never be forgotten that melancholic patients but too frequently refuse food, through some delusion or otherwise. My experience of a quarter of a century in treating the insane makes me concur with Blandford, that this is a most vital matter, and very often if twenty-four or forty-eight hours are allowed to elapse without *forced feeding* the patients will perish. The stomach-pump or nasal tube should be resorted to immediately, to avert such disastrous results.

Melancholia in women in many instances arises from, and is perpetuated by, menorrhagia and metrorrhagia. In some very interesting cases of this nature *repression of menstruation* has proved successful in my hands.*

* "Results of Repression of Menstruation," by E. O. Gehrung, M.D., American Gynæcological Transactions, 1889.

LECTURE XVI.

INSANITY—*continued*.—MANIA.—MONOMANIA (PARANOIA).—
DEMENTIA.—MORAL INSANITY.—IDIOCY.—IMBECILITY.

Acute Delirious Mania ("Typhomania"), or Delirium Grave—Treatment—Mania: its Characteristics, Course, Prognosis, and Treatment—Monomania ("Paranoia")—Dementia, Acute and Chronic—Moral Insanity: Diagnosis of; Illustrative Case—Idiocy—Moral Imbecility—"Linear Craniotomy in Microcephalia"—"Katatonia"—Hysterical Insanity—Transitory Insanity—Alcoholic Insanity—Insanity and Bright's Disease—Insanity following Influenza—Insanity complicating Heart-Disease.

GENTLEMEN,—In my last lecture I spoke to you about melancholia; to-night I begin with the discussion of a different variety of insanity, known as *mania*. For practical purposes we divide mania into *acute delirious mania*, *acute mania*, and *monomania*.

Acute delirious mania, or *delirium grave*, is that form of insanity in which maniacal symptoms exist with more or less elevation of temperature; in other words, when superadded to the ordinary symptoms of mania there is more or less heat, which can readily be detected by the thermometer. This acute delirious mania might more properly be called "*typhomania*," on account of the invariable existence of adynamic symptoms, but too often proving fatal. The symptoms are typhoid in character: the pulse is rapid and frequent, the tongue dry, and asthenia becomes more or less prominent. It is well to understand this condition, as it requires immediate and appropriate attention. Indeed, the disease often runs so rapid a course that there may not be sufficient time to effect the removal of the patient to an asylum. It is a matter of life and death, and, unless earnest measures are resorted to, the patient rapidly grows worse and sinks, which indeed is usually the case in spite of all our efforts to the contrary.

In acute delirious mania there is an active delirium accompanying the symptoms of mania not unlike what we meet with in acute febrile diseases, especially in typhoid fever. The delirium which is so prominent a symptom in certain low forms of fever

should not be mistaken for that of the disease under consideration, as the manifestations of the latter are much more active, and the absence of enteric complications constitutes a marked feature. Although the thermometer may run up to 105° F., the pulse be frequent and rapid, and sordes accumulate upon the teeth, if on your guard you can nearly always make a correct diagnosis.

The onset of the disease is usually violent and sudden, and it runs its course in a short time, sometimes lasting three or four days, at other times a week or longer. The aspect of the patient, history of the case, and peculiarities of the delirium will enable you to distinguish this affection from delirium tremens, meningitis, or ordinary cases of mania. The presence of tremor, peculiar visual hallucinations, and good-natured delirium in delirium tremens, the different character of the symptoms in meningitis, and the absence of fever and asthenic symptoms in mania, will enable you to avoid errors of diagnosis.

Spitzka, quoting Jessen, states, "In one of Jessen's patients anæsthesia became so extreme that he gnawed off the ungual phalanx of one of his fingers. That author claims that pemphigus-like vesicles appear in the otherwise apparently healthy skin, especially of the dorsal faces of the hands and feet. He believes this to be a comparatively constant sign; it was absent in two out of the five cases observed by the writer, while phlegmons and spontaneous gangrene were additionally noticed in one of the cases that had pemphigus. Most of these conditions are due to the vaso-motor paresis which marks this period."

Spitzka says, "The majority of the patients affected with grave delirium die in the delirious period after an illness of a few weeks; in those who do not die at this period the excitement continues unabated for four or five weeks, the subsequent symptoms of stupor increase, and the history closes with a fatal coma. *Complete recovery never occurs*;"* in rare instances the patients emerge from this severe disorder with a slight mental defect; in others paretic and terminal dementia supervene."

We should use stimulants freely, give nutritious, supporting food, and by all means induce sleep, always absent and very difficult to secure. Obstinate insomnia is found in all forms of

* Italics my own.

mania, and is frequently a pertinacious symptom. Of all the therapeutic measures used to promote sleep under such circumstances, I formerly believed that the most efficient was the *hydrate of chloral*. Of course it should be administered cautiously, and not be given in such large doses as were recommended by Sir James Y. Simpson; for, although chloral is an invaluable addition to the pharmacopœia, it is as potent for evil as for good, and care as well as judgment should be observed in its use. Sulfonal, however, is undoubtedly preferable to chloral; I regard it as the safest and most effective hypnotic with which we are armed.

It should not be forgotten that we must maintain the patient's strength by good and nutritious food of easy digestion and assimilation, and that concentrated liquid aliment, such as beef-tea, milk, alcohol, etc., is indispensable.

Very often persons afflicted with acute delirious mania become so violent that we are at a loss to know how to control them. One of the best measures to this end is to reduce the high temperature in the way recommended by Blandford and Sheppard, — *by the wet pack*. Dip a large sheet in *cold* water, and completely envelop the patient in it. After it has been on for an hour or an hour and a half, the patient having been wrapped in numerous heavy blankets, so as to induce free perspiration, the temperature will be found lower, and the active delirium relieved. Of course, care must be taken that the water be not too cold, that the application be not continued too long, nor the patient exposed to cold afterwards. Phenacetin, antipyrine, and quinine may be resorted to as antipyretics, but should be carefully watched.

Ordinary mania, such as you will generally meet with in asylums, as well as in private practice, may sometimes resemble "*acute delirious mania*;" but in the former there is no fever, neither is there a rapid pulse or a heated skin. Ideational insanity, generally preceded by affective insanity, exists in both.

Spitzka remarks in this connection, "*The morbid anatomy* of this disease consists in an intense hyperæmia of the brain and meninges. This is constantly found in patients dying in the excited period of the disorder; in those who die in the stuporous period the hyperæmia is sometimes obliterated by a collateral œdema; but in all the brain appears swollen, the cortical ganglionic elements are granular or opaque, stain poorly, and their periganglionic spaces,

like the adventitial lymph-sheaths, are literally crammed with the formal elements of the blood. In the single case examined *post mortem* by the writer white streaks were found on either side of the larger vessels in the pia. Microscopic examination showed that they were due to an accumulation of leucocytes, whose preponderance suggests an inflammatory nature of the lesion, rather than the condition of venous engorgement claimed by Krafft-Ebing. A most positive sign of inflammation was found in the case referred to: the arterioles were surrounded by an area staining in carmine with a beautiful pink flush, probably the expression of a molecular infiltration, while layers of newly-formed fibrin were found in and around the adventitia.

"That grave delirium is the result of a vaso-motor overstrain analogous to that supposed to exist in parietic dementia is supported by the etiology, the manner of origin, and the somatic sequelæ of this disorder."

MANIA.

According to Clouston, "Acute mania—the 'raving madness' of the older authors—is perhaps the type of all insanity, both in the popular and professional mind. Standing thus, and being the least rational, least conscious, most noisy, most unmanageable, and sometimes the most dangerous variety of mental disease, it affected the conceptions and the treatment of all other varieties in a most unfavorable way. In it many patients had no more 'reasoning power than a wild beast,' and all persons concluded to be insane (the conception of insanity was then a much narrower one, embracing much fewer persons) were accordingly treated by manacles and chains, stripes and darkness. Small compassion was felt for them, few laws protected them, little medical skill or study was exercised in their behalf, for they were reckoned beyond the pale of ordinary humanity. Even in Esquirol's time, at the beginning of this century, such patients are pictured in wild contortion and fury of look and action, and are represented heavily bound even in his illustrations. Yet this is a type of disease that is nowadays not at all so common as others. . . . Acute mania may be defined as intense mental exaltation with great excitement, complete loss of self-control, with sometimes absolute incoherence of speech and loss of consciousness and memory. After twelve

months it is arbitrarily no longer reckoned acute but chronic mania. Some authors set up a period of forty days during which alone the disease was to be called acute mania. This had no foundation in any clinical fact.

"Acute mania begins in various ways. The most common is by its commencing as simple mania and then passing into the acute form. But I have seen it begin quite suddenly, the patient being one hour a sane, rational, responsible being, and the next acutely maniacal. It has often a melancholic prelude. It sometimes begins by the patient expressing a delusion out of which, as it were, the extravagances seem to arise. Sometimes it begins by emotional, sometimes by intellectual exaltations and perversions, sometimes by both. At other times it begins by alterations of habit, appetite, and propensity. It commonly has premonitory symptoms, bodily and mental, such as headaches, a confused feeling in the head, a muscular fidgetiveness, an unrest of body and mind, a feeling that something is going wrong or something dreadful is to happen, a feeling of wild commotion in the head, as if it were about to burst, an impulsive desire to do something, to break glass or to do violence to those within reach. There are usually disturbed sleep and constant dreaming, usually of an unpleasant kind. I have known the temperature to rise to over one hundred before the patient could be said to be in any way maniacal.

"Krafft-Ebing has given a series of interesting clinical lectures upon this subject. He defines mania as a psycho-neurosis characterized by well-marked discordance, exalted sensation of impressions,—an abnormal sequence of psychological phenomena. Krafft-Ebing's theory of the brain-condition in mania is of hypersemia. He says, 'I think there can be little doubt that in mania we have a supernutrition with an overheating of the psychical machine.'

"He divides mania into two sections, designating them *mania mitis* and *mania gravis*, suggesting as synonymes maniacal exaltation and madness." *

What, now, are the *characteristics of mania* by which we may be enabled to diagnose it without difficulty? All cases of mania

* Brush, Annual of the Universal Medical Sciences, Sajous, 1891.

present more or less excitement,—mental excitement,—evinced by incoherent conversation, showing an irrational state of the mental faculties and a morbid association of ideas, to which are added wild gesticulations and almost constant motion. Maniacs dance, sing, and jump without adequate cause; their actions are violent and excited, their fury quite beyond their own control. These are the usual symptoms, the ideational insanity being plainly shown by the conversation of such patients; but there is, nevertheless, a perversion of the affective life underlying the disease and deeply impressing all its manifestations. This necessarily displays itself in action, and the patients are restless and destructive. They are difficult to manage, and their volubility is truly astonishing. This fact caused the older writers to contend that in maniacs the memory, as well as all the other mental faculties, became enhanced or brightened. Although such people show remarkable mental activity, “a complete change of emotional state, thus often becoming very joyous; a rapid and uncontrolled passing of the ideas through the mind; vivid kaleidoscopic mental pictures of the past; a tendency to constant talking, whether any one is present or not; passing from one thing to another, and soon becoming incoherent of speech;” speaking not infrequently in rhyme with astonishing facility, still these are no proofs of the above assertion, but simply point to abnormal increase of functional activity, resulting from disease. The mental mechanism is deranged. There is a defect in intellectual co-ordination, due to the absence of a controlling power. Not only is co-ordination necessary to the regularity and precision of muscular action, but mental action must also be governed by the same regulating influence, and in the cerebral hemispheres such inhibitory centres undoubtedly exist. As M. Allen Starr, in his “Familiar Forms of Nervous Diseases,” very aptly remarks, “Thought does not lead immediately to expression in all cases. Expression may be restrained: the impulse may be arrested. This restraint of the flow of thought outward in expression has been termed *inhibition*; and inhibition, or the act of control, is the highest of all the cortical functions.” When this inhibitory influence fails to act, or acts imperfectly, there is, figuratively speaking, a loosening of some contact, in electrical parlance, or some lever is out of order; and, though the machine may be in rapid and continuous motion, with a

striking evolution of thought and brilliant scintillations of fancy, still, the ideational workings are abnormally performed, and the intellect is impaired. We have here a condition of irritability of the brain, the same that exists in hyperæmia, or in inflammatory conditions of the brain-envelopes. You remember that when speaking of meningeal inflammation I told you that the symptoms of irritation preceded those of depression. So we have in mania a certain hyperæsthetic state of the nerves of special sense, and of the perceptive and psychical powers, giving rise to the symptoms of irritation, whose pathology is very similar to that of congestive diseases. There is, of course, an increased flow of ideas, the mental powers are acting abnormally, and there is a greater scope, though less perfection of activity, which corresponds with the concomitant symptoms of irritation. "There may be ceaseless laughing, or scolding, or swearing; conversations are held in loud tones with imaginary people whose voices are sometimes heard or their forms seen. Sometimes there is rhythmic action of mental and muscular centres seen, evinced by rhyming all the ordinary conversation, or by regular movements of the limbs and body." But, as the malady progresses, the patient is finally completely lost in the labyrinth of mental disease, and dementia appears, a state corresponding with the symptoms of depression of meningitis, and fatuity may be anticipated. In maniacal excitement, and the dementia which follows, there is at first an increase and then a decrease of mental activity, invariably accompanied by very imperfect performance of the intellectual and moral functions. "Conversations with old friends now dead will be carried on. Scenes of childhood and years gone by will be vividly realized. Self-control may be utterly lost; the temperature over 99°; the pulse quick and sometimes full; the skin moist; the tongue furred; the appetite gone; the tastes and sense of decorum and decency perverted." (Clouston.)

To illustrate this, suppose the case of a man who in rowing a boat, instead of making slow and regular strokes, lacks the necessary co-ordinating power of muscular action, and makes violent and spasmodic efforts, which necessitate a considerable though inadequate and improperly regulated expenditure of strength; his exertions effect but little; many of his strokes would hardly equal one regular and natural sweep of the oars. In mania there may

be more mental activity, but its vigor, far from being increased, is diminished.

The most prominent symptom of mania is the mental excitement, and, correspondingly, we always find an exaggerated feeling of self-esteem and self-exaltation. The patient, in his own estimation, is better than anybody else; and there always exists a presumptive superiority of some kind. Schroeder van der Kolk holds this to be an indispensable symptom. Obstinate insomnia, especially in the earlier stages, is rarely absent. With this fact all physicians treating insanity are familiar. Troublesome constipation is usual. A remarkable tendency to the manifestation of thought by action is characteristic of mania. Certain actions, representing thoughts, almost simultaneously accompany their utterance in words. Such patients therefore gesticulate violently, and act while speaking. The impulse to incessant motion is truly remarkable, and sometimes quite dangerous, a fact which is interesting in a medico-legal point of view. "Thought is father to the impulse;" but here the thought is hardly recognized before the muscular action gives it expression. It is in consequence of this that such patients run, dance, sing, kill, and destroy. Because of this tendency to the exhibition of thought in external action they are in constant mischief and trouble. I hope you will all seize the important distinction, that when the violence of action manifests itself by suicide, homicide, etc., such is not the only expression of the insanity; other well-marked symptoms will be found to coexist. Dangerous propensities alone cannot correctly define distinct forms of insanity. On these grounds I exclude pyromania, erotomania, dipsomania, homicidal and suicidal mania, as types of insanity, simply because they embrace only a single sign or symptomatic indication of the existence of unmistakable insanity; just as cough does not constitute pneumonia or bronchitis, but is only one of its accompaniments.

The course of mania is very uncertain; it may last a week, a month, or a lifetime. The tendency of the disease is either towards a favorable termination or to chronicity; or it may result in dementia,—the "tomb of reason:" once herein engulfed, man is no longer a rational creature, and that divine attribute which distinguishes him from the animal is lost forever.

It is of course advisable to treat the disease in the early or

inceptive stage when possible. The moral treatment is as indispensable in mania as it is in melancholia, and hence asylum discipline should be resorted to early. Mania, to some extent at least, seems to yield to therapeutic measures, and the bromides of potassium, calcium, sodium, and lithium have been employed with advantage, on account of their sedative powers, and their controlling influence over the circulation of the brain. When we come to study the pathology of insanity, we shall see that there may be not only too much blood in the brain, but also too little, and that the blood may be abnormal in quality as well as in quantity, resulting in "*irritable weakness*." It follows that while bromide of potassium may be serviceable in some cases of mania, in others it will be productive of harm; as, for instance, when an anæmic condition of the brain exists.

The insomnia must be combated. I have already recommended hydrate of chloral; but I consider sulfonal to be the hypnotic *par excellence*. Free pustulation of the head, by the application of croton oil to the scalp, often acts marvellously in relieving the patient, especially when the disease is becoming sub-acute in character. Sulphate of copper, ergot, and Indian hemp, combined with the bromides, as recommended by a distinguished foreign authority, cold affusion and tepid baths, cautiously administered, the hypodermic injection of morphine and hyoscyamus (often exceedingly efficient), are remedies which have all proved useful in my hands for the treatment of intense maniacal excitement. When the motor centres are seriously implicated, I believe that conium is a most useful remedy, and will produce striking results in quieting the restlessness, jactitation, and general excitement. As tonics in subsequent stages, I have found none equal to cod-liver oil, dilute phosphoric acid, quinine, and the preparations of iron. In mania, a *cautious* hypodermic injection of sulphate of hyoscyamine (hydrobromate of hyoscine I do not consider safe) is one of the best hypnotics, and hydrobromate of conine one of the best agents to quiet motor excitement that I have ever used.

The hygienic conditions should not be neglected. Let us never forget that the excitement is accompanied by excessive retrograde metamorphosis of tissue, and keeps pace with these destructive changes. In acute mania, therefore, you should always admin-

ister large quantities of nutriment ; and, where the patient *resists*, it is your duty to compel him to take it, even, if you must, by the stomach-pump. Sudden death often occurs from intense excitement, when followed by corresponding depression and exhaustion. Whenever very intense activity exists, whether functional or organic, you have reason to dread the consequent reaction.

The prognosis depends greatly upon the duration of the disease, the age of the patient, and the number of previous attacks. If the family history point to insanity, you may reasonably fear a relapse, and hence will give a guarded opinion ; one attack of insanity always predisposes to another, and this more particularly when the patient is of an insane temperament.

MONOMANIA (PARANOIA).

What is monomania ? It is a partial, a delusional form of insanity ; and the name is derived from the Greek words *μόνος* (single), and *μανία* (mania), as it was supposed to be an insanity upon one particular point, or a disease of only one portion of the brain. In monomania the intellect is apparently clear, or even unusual mental activity is at times exhibited, except upon a single point, or perhaps a few points, indicative of the patient's insanity. This form has also been called *delusional insanity*, because the disease is always expressed by one or more delusions. Overweening self-esteem exists in monomania even to a greater degree than in mania. It is an almost invariable concomitant of the disease, and I have never seen a case in which it did not constitute a well-marked feature. Such persons grow exceedingly angry if you differ with them in opinion. They are often very intelligent, and may astound you by the acuteness of their reasoning powers, their general information, and their brilliancy of thought.

Are we to understand that such a thing can really exist as a *partial* insanity ? Can an individual be *non compos mentis* to a limited degree and mentally sound otherwise ? Can a truly definite pathological condition of the mind ever be truly partial ? Can a man be insane in one or two ideas and mentally sound in all others ? I am not inclined to believe such a doctrine, but prefer to think that in this affection the mind is more or less completely impressed or biassed by the delusion or group of delusions. It does not follow that because a man is seemingly insane upon

but one subject he is sane in all other respects, since we cannot measure the extent of his irrationality, or positively ascertain the exact limits of his unsoundness. We cannot restrict disease of the mind by any arbitrary barrier. I cannot conceive of the existence of a delusion unless a greater or less implication of some, if not of all, of the mental faculties coexists as a result thereof. This may not be apparent on examination, but for all practical purposes we can accept it without hesitation.

By way of illustration, I may remark that I once knew a man who was on trial for some crime: he was examined by a committee of experts, who, after a careful investigation, could find no evidence of insanity. A relation of the accused, however, requested his attorney to ask him "why he was more powerful than other men." His manner immediately changed; and he replied, with great vehemence, that he "held his power from the source of all glory, and was on terms of social equality with the Holy Ghost, and with other members of the Trinity, being in the habit of treating them to the best of liquors." Men will labor under such delusions, believing them as firmly as you would believe a mathematical fact. All the regions of the mind are, to some extent, pervaded by such delusions, when they exist; and a man in such a condition must, to some extent, be mentally unsound in every other respect. A person laboring under monomania, though apparently sane in many particulars, is, nevertheless, entirely *non compos mentis*, and ought, in most instances, to be secluded in a lunatic asylum; for when subjected to the soothing influences of such a retreat he may cause little or no trouble, but if brought in contact with the world, where he would be liable to be crossed, he might become dangerous, or even perpetrate some fearful deed.

It is a fact of special pathological significance that this disorder is rarely primary. The anamnesis generally shows a previous attack of mania, possibly of melancholia. The patient has been apparently cured, having become comparatively rational, with the exception perhaps of some one lingering delusion, from which he never recovers. I have had more than twenty-five years' experience in the treatment of insanity, and do not know of a single case of cure of monomania. In cases of apparent cure there has been a cessation of the previous primary pathological processes, consisting

simply in a diminution of their intensity and perhaps of their extent. Monomania does not commence as such by any primary pathological change. We know that insanity is always a disease of the brain, but monomania is generally the result of some other form of insanity, in the convalescence from which there has been a clearing up of the morbid condition to a certain point, at which it is arrested, and thus monomania remains for life.

PARANOIA.

This term, which so frequently occurs (Spitzka classifies paranoia with monomania) in the psychopathic literature of the day, is defined by Billings, in his "National Medical Dictionary," as "unsoundness of mind, crankiness, insane diathesis, hereditary or acquired chronic mental instability; the 'protopathic insanity' of Bucknill and Tuke. Sometimes used to signify monomania with delusions."

M. Allen Starr, in his recent work on "Familiar Forms of Nervous Disease," states that "in most of the cases of paranoia a hereditary history was readily elicited. All had either fixed systematized persecutory delusions alone, or these in combination with delusions of a grandiose character." "A typical case of paranoia exhibits certain positive features which make this form of insanity a clinical syndrome. There are hereditary taints; some eccentricities in childhood; more marked peculiarities during youth, often associated with a degree of hypochondriasis; and at about the age of thirty, sometimes earlier, sometimes later, the growth and systematization of delusions of *persecution*, which may in turn be combined with or give place to systematized delusions of an exalted character (either religious, philosophic, patriotic, or erotic). Such delusions completely dominate their entire mental action without impairing every faculty."

These cases were formerly called "monomania" by American authorities, and are classified as "monomania" or "delusional insanity" by nearly all the English writers.

As M. Allen Starr further observes, "The eccentric individuals known as 'cranks' are without doubt imperfectly developed cases of paranoia."

In conclusion, Starr continues, "As regards the treatment of these patients, little can be done except perhaps in the way of

moral methods, and these are of most value in the earliest stages. They may abort the psychosis. The asylums are the destination of most paranoiacs, and in the best of these institutions the discipline, employment, recreations, and regularity of eating and sleeping exercise a beneficial influence upon the course of the disease.

"Paranoiacs should be very carefully examined, especially those with *persecutory* delusions, as to the presence of ideas of retaliation and vengeance upon their persecutors. If there is any suspicion of their possessing dangerous tendencies, they should, of course, be deprived of their liberty as soon as possible."

The next form of insanity to which I desire to call your attention is

DEMENTIA.

I have very little to say concerning this affection, having already spoken of it as "the tomb of reason." Some speak of *acute dementia*, as distinguished from *secondary dementia*. By acute dementia is meant that condition which sometimes follows severe fevers, moral shocks, or physical injuries. In these instances the mind, for a time, becomes a perfect blank. I will illustrate this by a case which Professor Dickson, of Philadelphia, was accustomed to relate to his class. A very eminent and erudite divine, of New Jersey, suffered from a severe attack of typhoid fever, from the immediate effects of which he recovered, but his mind was completely wrecked. He had forgotten everything,—could write or speak upon no subject, having ceased to remember not only the elements of arithmetic, but even the alphabet. Being an industrious man, and possessing great tenacity of purpose, he was not overcome by his calamity, but immediately commenced to study energetically, in order to acquire the rudiments of an ordinary education, and thus regain, if possible, all he had lost. One morning, after months had elapsed, the darkness and obscuring clouds with which his mind had been surrounded were suddenly dispersed, the light dawned upon him, and he found himself possessed of his memory once more. It was like the reappearance of the sun after temporary obscuration by a passing cloud.

Asphyxia may have the same result. I remember being once asked by a lawyer, during a cross-examination, a question very pertinent to the subject we are now studying. A gas com-

pany had been sued for damages on account of negligently allowing the escape of gas, which was alleged to have produced acute dementia in an employee, who had been resuscitated from a dangerous state of asphyxia. It was contended by the defence that gas could not have had such an effect, and that the insanity must have pre-existed. The attorney for the company, desirous of showing the similarity of effects between the different forms of asphyxia, many of which he contended were very commonly known and had never been supposed to result in insanity, asked whether I had ever heard of strangulation resulting in mental impairment. I replied in the affirmative. In any variety of strangulation the blood becomes super-carbonized, and necessarily produces deleterious changes in the brain, which may result in some form or other of insanity, perhaps more frequently in acute dementia, damaging the individual very seriously. The lawyer, desiring to propound a perplexing and, as he supposed, absurd question, inquired, "Suppose, doctor, that a man be almost drowned, just saved in time to be revived: do you mean to say that this might result in grave mental impairment?" "Why, of course," I replied: "you have furnished me with an excellent illustration, demonstrating how asphyxia may result in acute dementia independently of what has caused the asphyxia." This shows the importance of being able to turn their own weapons against those who point them; for many lawyers find no little satisfaction in making medical men appear ridiculous when called upon the witness-stand.

By chronic or secondary or terminal dementia is meant that form which follows the acute varieties of insanity, either mania or melancholia, etc. When you pass through the wards of an insane asylum, you will recognize such patients by their blank countenances. There is an entire absence of intellect, and an incapacity, more or less developed, for performing any rational action. You will, perhaps, see them fondly nursing a stick of wood, believing it to be a favorite child, or in the active pursuit of some delusion; in fact, they are reduced to perfect imbecility, and this disease is the gulf into which the various forms of insanity may drift. Dementia presents the traces of violent precursory storms, which have stranded the nobler faculties of man, thus animalizing him. Many have not the instincts of

beasts, their lives being purely vegetative. Here again we see the necessity for a proper treatment of acute insanity ; for what can be more terrible than this condition of abject mental degradation ? The culpable neglect of the physician in not sending such unfortunate beings, while yet in a curable condition, to an asylum, entails upon them a life of misery. When sent to an asylum they are but too often already hopelessly demented, and reduced to a condition which is horrible to witness. They are so filthy as often to eat their own excrements ; and it will excite your sympathy to remember that these people were once rational beings like yourselves, and that their fate might have been, at least, partly averted by timely assistance.

The following quotations concerning *dementia* are made from Savage's recent work on "Insanity and Allied Neuroses :"

"In considering dementia I shall make two clear divisions. In one there is destruction, more or less complete, of the mind, which can never be recovered from, and in the other there is functional arrest, which may pass off. . . .

"As mental life begins with but little evidence of intellect, and with imperfectly organized sense-impressions and motor impulses, so it may end with a return to its simplicity in age. At the one end of life there may be inability to develop intellectually ; this is called *amentia* ; and at the other end destruction of mind may leave the whole intellectual fabric a ruin ; this is called *dementia*. . . .

"No two houses fall into ruins in exactly the same way, though in the end the four walls alone may remain as evidence of the once-inhabited dwelling ; and so with mental destruction, it will be found that, though in the end similar foundations and simple boundaries of mind may remain, all the finer parts are removed : whether age, war, or fire has destroyed the houses, the results are alike ; similarly, either age, disease, or injury may wreck the mind. It will be seen that the mind may show the effects of destruction in various ways, and the destruction may progress at very different rates. There is no such thing as complete dementia, for life could not exist with total suppression of mind and sense-reaction. . . .

"Dementia is divided into *primary* and *secondary*.

"In complete general dementia there exists a general weakness of the senses, the memory, and the higher organizing and con-

trolling power. The senses react slowly to their respective stimuli, reflex actions are performed, and in some cases the loss of the higher control causes reflex action to be rendered more rapid and more active than in health.

"Many acts are done automatically. The power of storing impressions is greatly impaired, or even annihilated, so that the memory for recent impressions is wanting, and memory of the past is somewhat affected. There is no evidence of volition, and emotional display is rare. The loss of self- and general control is marked. . . .

"Of course the mental faculties are nearly abolished.

"Appetite and digestion may be good, but the patients are usually dirty and neglectful in their habits. They usually sleep well. Persons thus afflicted generally have to be fed, washed, and cared for like children.

"Dementia may be due to either physical or mental disease. Among the purely *physical* causes, fevers very frequently produce it, especially typhoid fever, which is known not infrequently to seriously impair the memory for a longer or shorter period. Alcoholic and other excesses, rheumatism, syphilis, epilepsy, child-bearing, blows on the head, pneumonia, previous attacks of mental disorders, are all well-known factors in the production of dementia."

MORAL INSANITY.

What is moral insanity? Is it an insanity of a man's morals? Is it that condition in which an individual has impairment of mind, destroying his knowledge of right and wrong, and by which he is led to commit nefarious deeds? Is such moral insanity? It is very important to understand this subject, as it is often constituted a plea in legal cases. It is never moral depravity, and moral depravity is not always moral insanity. If you realize this, you have made an important step in advance. In a previous lecture, when I cited Blandford's illustration of an Italian brigand, accustomed to kill and plunder, I showed you distinctly that this was not moral insanity, but only a blunting of the conscience by the habitual commission of crime. I maintain that moral insanity is but a variety, perhaps a peculiar form of the affective type of insanity, of which you have lately heard so much. Affective, impulsive, pathetic, emotional, and moral insanity are virtually

the same, each and all of them belonging to the same type, as they all relate to *action*. Pritchard, who first described moral insanity, attached great importance to it, especially in a medico-legal point of view. He maintained that in moral insanity there was no evidence, to any degree, of intellectual impairment or implication, and that the judgment, memory, cognition, and perceptive powers were normal, but that there was a perversion of the moral faculties, resulting in a change of the habits, feelings, affections, propensities, and sentiments of an individual, sometimes, though most rarely, accompanied by delusions, whereby he was rendered insane and irresponsible. He defines it, to use his own words, as "a morbid perversion of the natural feelings, affections, inclinations, habits, moral dispositions, and natural impulses, without any remarkable disorder or defect of the intellect or knowing or reasoning faculties, and particularly without any insane illusion or hallucination." I formerly believed this to be true, but am now satisfied that in every case related by Pritchard more or less mental weakness or impairment existed, discoverable had it been carefully sought for; and while I consider moral insanity to be a morbid perversion of the moral faculties, I am satisfied that every case is accompanied by more or less mental defect; in other words, in moral insanity more or less ideational insanity always exists, though it is not always apparent nor always readily discoverable.

Pinel, referring to this class of cases, states that moral insanity is largely a matter of bad education. Folsom observes that insanity of a purely moral character is "an uncontrollable violence of the emotions and instincts, and is probably as rare as purely intellectual insanity."

In another place the latter remarks that moral insanity "is recognized by all the authorities on mental disease, whatever may be their opinions as to the limitations of responsibility in it. It is especially to it that we can apply the words of the Autocrat of the Breakfast-Table, that the worst forms of insanity are those to which the asylum shuts its doors. It is marked by moral perversion, change of character and action, and so little intellectual impairment as to be easily overlooked by one not familiar with morbid mental phenomena."

We particularly agree with Folsom when he asserts that, "Although moral insanity is probably a common cause of young

persons of both sexes being led into lives of licentiousness, wickedness, and crime, it is to be carefully differentiated from deliberate yielding to temptation and following lives of vice until a strong enough motive is offered for doing better or a punishment is made sufficient to be deterrent. . . . Moral insanity is a defect in the affective sphere, but also an intellectual defect of a peculiar kind, which is often concealed under the mask of a perverted moral sense, and which requires time and practice on the part of the physician for its detection." (Westphal.)

As to diagnosis, I must say that I never accept the theory of moral insanity without certain corroborative antecedents of some other form of insanity,—some evidence of the insane temperament, or at least of a strong taint of insanity in the ancestry, while I also seek other important links in the history when obtainable; especially as *the present weight of authority is negative* as regards its existence, and since, moreover, it is not generally recognized by the courts. A change in the individual's self is a most important symptomatic manifestation: without this as a basis we cannot possibly affirm its existence. There must be a change of character, "without external adequate cause," not explicable by the ordinary motives of human actions. Suppose a man is waylaid and killed by some miscreant, the money upon his person stolen, and that the thief when prosecuted should enter a plea of moral insanity with design to defeat the law: would not the circumstances and the motive be sufficient to warrant an expert in insanity in unhesitatingly giving his testimony against such an assumption? But, on the other hand, suppose an upright, honest, moral man, known for a lifetime to be conscientious and above reproach in all his relations, *suddenly*, without *motive*, become obscene and lascivious in his conduct, evincing murderous and thieving propensities: if there be superadded an insane ancestry, temperament, or predisposition, we have certainly good evidence of moral insanity. If, however, moral insanity exist, and such a case be investigated, a certain amount of mental weakness will be usually found, pointing to more or less ideational insanity as a complication. Under such a limitation only can I admit the existence of moral insanity, but never *per se*, or without some degree of actual mental impairment.

"But Dr. Tuke has now furnished the advocates of moral

insanity with an argument which legal scepticism will find some difficulty in answering. Skilfully abandoning the old definition as untenable, this eminent alienist contends that the existence of moral insanity is proved by the production of cases in which (a) disorder of the moral faculties is *the prominent characteristic*, or (b) there is at least no such intellectual unsoundness as the *law* would admit to be an exculpatory plea.

"But Dr. Tuke has not contented himself with maintaining that an entire absence of mental disorder is not a necessary *factum probandum* in the case for moral insanity. He has placed on record, temperately, clearly, and with ample detail, at least one case of congenital moral defect in which no lesion of the intellectual faculties appears to have existed. To transcribe the history of this case would be unfair to the author, and no *précis* of the facts would bring home to your minds their full significance. I shall conclude this brief communication by expressing my firm belief that Dr. Tuke's *observation* will be the 'locus classicus' of moral insanity; that it justifies the cautious acceptance of that doctrine by which American judges have honorably distinguished themselves; that it is worthy of consideration both by medical experts and by those whose duty it is to cross-examine them, and that the possibility of '*primäre Verrücktheit*' has been established at last."*

We have now to consider two other forms of insanity:

IDIOCY AND IMBECILITY.

By the former is meant that form of insanity which is the result of an arrest of development or growth of the brain either during foetal life or immediately after birth. Imbecility differs from this only in degree. In it the arrest of mental development occurs at a period subsequent to birth, often following some pathological process. In fact, idiocy is an original defect in the organization of the brain, while in imbecility the defect is adventitious, for the physical condition of the brain may have been perfectly normal at birth, or even at a subsequent period. Still, it is as difficult to draw a line of distinction between idiocy and imbecility as between insanity and reason.

* A. Wood Renton, of the London Bar, *Medico-Legal Journal*, New York, September, 1891.

(Packard.)

"This is perhaps the most appropriate place in which to mention a striking instance of the extension of the province of surgery in a new operation, to which has been affixed the name of *craniectomy*. (This term, according to analogy, should signify removal of the skull by a cutting operation; *linear craniotomy* would be more accurately descriptive of the procedure.) The condition which it is proposed to relieve, and which has been thus treated in several cases, is that known as microcephalus, or, more properly, microcephalia.

"The idea is that the resistance of the imperfectly developed or prematurely solidified skull checks and stunts the natural growth of the brain, and children so affected are apt to be idiotic in a greater or less degree. Hitherto such cases have been regarded as beyond the reach of medical or surgical treatment, and as susceptible only of such slight improvement as might be brought about by patient efforts and training. It is now proposed to remove one of the factors in the arrest of development by relieving the brain of mechanical compression, thus affording it opportunity to expand.

"Guéniot claims to have made the original suggestion of surgically relieving the brain of the pressure of a too small cranium, in a communication made to the Académie de Médecine in November, 1889; but Lannelongue reported two cases in which he had carried out the idea with encouraging success, and Keen has operated in nearly a similar manner, the result of the cases, however, being not yet placed upon record. All of these cases were in children, and the condition was congenital. But it should be noted that Bauer (of St. Louis) mentions a case in which he trephined a man, aged twenty-seven years, for acquired microcephalia from an injury of the skull sustained sixteen years previously; very great improvement followed, the patient becoming able to go into business, and when seen six years later being in full health and mentally sound. Bauer reports that another case, that of 'a young woman' (age not stated), in whom the condition was probably congenital, was operated on recently by him with some benefit: the procedure consisted in trephining at two points and removing the bridge of bone, the right parietal being first attacked, and the left after an interval of about a month.

"The operations performed by Lannelongue and by Keen were more extensive, so as to give ample room for expansion, the side of the head being made into a bony flap, attached below and free above. Lannelongue contented himself with dealing with one side only, the left; Keen operated on the right side, proposing to attack the other side later if the circumstances should seem to indicate such a course. . . . For cosmetic reasons, Keen made his incision of the skin entirely within the limits of the hairy scalp. . . . It seems likely that the initiative thus given will be followed in other cases. I have had the privilege of witnessing two of Keen's operations, and the procedure is certainly a brilliant one, of surprising ease in skilled hands." *

This summer I treated a young child from Texas for traumatic epilepsy. At the time of my first examination of the patient, then between six and seven years of age, I found an ugly scar on the left side situated over the motor area, near the root of the ascending frontal convolution.

At the age of two and a half the child sustained a serious fall, of from ten to fifteen feet, striking its head upon some loose bricks at the point where I found the scar. The boy was a magnificent specimen of physical development, and prior to the traumatism was learning rapidly to speak and showed unusual intelligence. The mother told me that, although she had several other children who were quite intelligent, our little patient was before the fall the brightest member of the family. Immediately after that, however, convulsive epilepsy without focal symptoms developed, and the child became deaf and mute. All hope was abandoned, after protracted treatment by many eminent physicians.

In consultation with my friend Dr. H. Tuholske, professor of surgery in the Missouri Medical College, an exploratory operation over the site of the scar was determined upon. After turning back the scalp we found extensive ossification of the cranial sutures, and the operation of trephining was abandoned. A month later we decided upon *linear craniotomy*. This operation was performed in December, 1891. The child passed safely through the dangers attendant upon the operation, but sufficient time has not yet elapsed (January 15, 1892) for the observation of any definite results.

* Annual of the Universal Medical Sciences, Sajous, 1891.

I desire to report in this place another case occurring in my practice which was operated upon on November 14, 1891, by Dr. Tuholske. The details are briefly as follows. Parental history good; first and only child; anterior fontanelle small at birth and entirely closed at six months. The child did not use its hands like other babies, could not sit or hold up the head. The wrists were constantly flexed. Sight and hearing were good. There was no attempt to speak; imbecility. Operated on when past two years. During the operation the chisel was abandoned for Keen's rongeur-forceps, on account of pronounced symptoms of concussion. Healing by first intention; left hospital two weeks after operation. Dr. Tuholske tells me he has lately heard from the mother in a letter dated January 6, 1892. She reports that marked mental and physical improvement is already declared, and gives details of the child's condition showing this to be the case.

I must tell you that microcephalia is frequently due to a too abundant supply of lime salts, as where drinking-water is highly charged with lime, or where too much lime-water is added to the milk of bottle-fed children; perhaps, indeed, it is almost always due to some such cause, except where chronic inflammation is plainly the originating condition.

One more form of insanity to be considered is

MORAL IMBECILITY.

In mental imbecility the actions express a want of activity of the higher intellect, but in moral imbecility there is an absence of the manifestations of the moral intellect. This is an interesting psychological condition.

Have you never at school seen boys expelled who had no idea of moral obligation, conscience, or the distinctions between *meum* and *tuum*? who were constantly in "hot water," and a source of anxiety to their parents? in other respects intelligent scholars, often showing mathematical or mechanical talent, but always lying or stealing, and furthermore given to voluptuous and intemperate tendencies and obscene habits? boys who would forge checks to obtain money; always in trouble while young, and under the eye of the police when grown; although belonging to good families, with every advantage of education and religious training, they were the black sheep in the fold. You cannot have been close

observers if you have not seen such cases, for in every community you may find numerous instances. A boy of this description will be as much trouble to the family physician as to any one else, as the parents will be unable to explain his actions, being averse to thinking him insane, on account of his being so bright in many respects.

Such is moral imbecility. Such people have the misfortune to be born and grow up without the development of those parts of the brain which preside over the elaboration of the moral faculties, and are hence a source of mortification to their relations and a curse to themselves.

Some authors contend that moral imbecility may be congenital, or may be acquired as a result of disease contracted or injuries received during childhood. My friend Dr. William B. Hazard, now deceased, once related to me the case of a boy who had always shown excellent moral tendencies until the age of twelve years, when he passed through a severe attack of typhoid fever; after his recovery there was no impairment of the intellectual faculties, but there was such a decided alteration in his morals that his parents were firmly convinced that during his sickness he had become the victim of demoniacal possession.

KATATONIA.

Spitzka defines katatonia "as a form of insanity characterized by pathological emotional state and verbigeration, combined with a condition of motor tension."

It was first described by Kahlbaum, of Görlitz, and has been more recently studied by Spitzka and Kiernan.

It usually commences with symptoms resembling melancholia, which are followed "by a period in which the patient presents an almost cyclical alternation of atony, excitement of a peculiar type, confusion, and depression, which finally merge into a state of mental weakness, approaching if not reaching the degree of a terminal dementia. Any single one of these enumerated phases may be absent."

"The prognosis of katatonia is relatively favorable as regards life, although the danger of pulmonary tuberculosis developing in the depressed and atonic stages of the trouble is not to be lost sight of." (Spitzka.)

Folsom states that in katatonia "there is an underlying well-marked intellectual impairment, slowly advancing in incurable cases to pronounced dementia. Delusions, more of the unsystematized than of the systematized character, but resembling both, constitute a prominent part of the disease from the beginning. Verbigeration and a curious sort of pomposity are usually found in more or less pronounced degree. The delusions are mixed. They are exalted, hypochondriacal, melancholic, with all sorts of self-accusation, and may be full of suspicion, fears of poisoning, and ideas of persecution. Hallucinations of the special senses and illusions are not uncommon. If the term katatonia is not used, or at least if a special place in the nosology were not given this disease, it would be difficult to know whether to class these cases as primary dementia, melancholia with delusions, delusional insanity, or confusional insanity."

"The verbigeration, when it exists, and the expression of delusions are often associated with a manner on the part of the patient suggesting disbelief in them, and sometimes the patient smiles or laughs at the astonishing character of his statements. There is a self-conscious element at times, suggesting mimicry or hysteria; a certain pathos is universal; opposition and contradiction, even to refusal to eat, leave the bed, dress, wash, are quite common; and nurse and physician are tired out with the monotony of the mental and physical state. Well-marked catalepsy is not common in my experience, although it occurs; and in all cases I have seen the mental state and physical atony suggesting that condition. Little attention has as yet been given to katatonia in asylums in this country. Judging from my own experience, it is not a common disease." (Folsom.)

Prolonged mental exhaustion and syphilis have been advanced as probable factors in the production of katatonia.

The course and duration of the disease are tedious, with tendency to relapse after apparent recovery.

It is probably a neurosis; its nature is quite obscure.

HYSTERICAL INSANITY.

Hysterical insanity is more frequent among women than among men. Hysterical symptoms, as I stated in a preceding lecture, may complicate any organic or functional disease of the nervous

system; and I regard it as very doubtful whether a distinctive form of insanity termed hysterical is entitled to a nosological position.

Folsom asserts that "it is characterized by extreme and rapid mobility of the mental symptoms,—amnesia, exhilaration, melancholic depression, theatrical display, suspicion, distrust, prejudice, a curious combination of truth and more or less unconscious deception, with periods of mental clearness and sound judgment which are often of greater degree than is common in their families; sleeplessness, distressing and grotesque hallucinations of sight, distortion and perversion of facts rather than definite delusions, visions, hyperæsthesias, anæsthesias, paræsthesias, exceeding sensitiveness to light, touch, and sound, morbid attachments, fanciful beliefs, and unhealthy imagination, abortive or sensational suicidal manœuvres, occasional outbursts of violence, a curious combination of unspeakable wretchedness alternating with joy, generosity and selfishness,—of gifts and graces on the one hand and exactions on the other. The mental instability is like a vane veered by every zephyr. The most trifling causes start a mental whirlwind. There is no disease giving rise to more genuine suffering or appealing more strongly for the sympathy which, freely given, only does harm. One such person in the house wears out and outlives one after another every healthy member of the family who is unwisely allowed to devote herself with conscientious zeal to the invalid."

The prognosis is unfavorable.

The treatment depends largely upon mental discipline and moral measures. All useless sympathy should be excluded. Diversion, the development of self-control, and general hygienic measures are matters of the most essential importance. Never was better professional advice given than that of Folsom when he states that "the temptation to use drugs is, like the fascination of being pitied and petted, very great, as alcohol, chloral, or opium often acts like magic for the time being, and there is a general craving for one or all of them. But they are utterly demoralizing in the end. The habitual use of stimulants and narcotics in such cases only increases the evil. The fact must be recognized that the hysterical insane are often least responsible where they seem most so, and that they must be treated with unending patience, kindness,

gentle firmness, and a wise ignoring of most of the symptoms." I have found from long experience that the salts of gold, as originally recommended by Niemeyer in hysteria, are very valuable.

Weir Mitchell's "rest-cure" will frequently produce happy results.

"Cases have been reported of two persons being exposed to the same causes and having similar attacks of insanity (*folie à deux*), and also of *folie induite* or *folie communiquée*, where several persons have adopted the delusions of a person of influence among them, one of the most remarkable instances of which was the case of the seventeen grammar-school-taught Adventists of Pocasset, who accepted the insane belief of their leader, Freeman, that, like Abraham, he had been commanded by the Lord to kill his child, that she was to rise on the third day after he had killed her, and that he was to become a great evangelist. So-called epidemic insanity, choreo-mania, and demonopathy belong in this class. . . .

"From untrained, ill-balanced men and women, whose lives are ill regulated, the ranks of the insane are largely filled. Insanity is often the ultimate wreck of a life ill guided, directed chiefly by caprice and passion, and weakened by indulgence. In that case it is, much like habitual drunkenness, as much a fault as a disease. The individual will not behave with decency and propriety for so long a time that finally, especially after the age when the brain begins to fail, he cannot." (Folsom.)

TRANSITORY INSANITY.

"*Transitory insanity* is used by Krafft-Ebing as indicating mental disease differing from other insanity only in the fact that it is of short duration,—namely, from two to six days. If it is applied to sudden and transient outbursts of mania, with delirium, loss of power of self-control, and inability to clearly recollect the circumstances of the attack and what happened during its continuance, it is a rare disease, occurring for the most part in epileptics and in persons under the influence of alcohol or addicted to its habitual use. It is sometimes under the latter-named condition called alcoholic trance. It consists in an automatic state resembling the epileptic delirium, which may occur also in sleep and resembles somnambulism. The actions are guided by co-ordinated

will without conscious intelligence, and may consist in crimes and brutalities and foolishness entirely inconsistent with the character in health. It seldom lasts more than a few hours. When caused by alcohol or as a symptom of epilepsy, it may occur without other marked inciting cause; otherwise it is commonly due to mental shock. Several cases happened during the mental excitement of the first battle in our civil war. The most striking case within my own experience was that of a man who under the strain of prolonged grief and the mental shock of a great fire destroying a large part of the town in which he lived, perhaps moderately affected by alcohol, suddenly grasped an axe and cut off with one blow the head of a beloved child. He was found in the street without knowing how he had got there or what he had done. One attack is the rule, although several, probably of an epileptic nature, have been reported. It is an extremely difficult condition to diagnose with certainty, and is therefore often the refuge of criminals and a resource of criminal lawyers. The most likely honest mistake liable to be made regarding it is to confound it with an outburst of passion." (Folsom.)

The above views, I must confess, are not altogether in accordance with my own impressions. All cases of transitory insanity, in my opinion, are of epileptic origin, and if the histories of the above cases were carefully scrutinized, I think it almost certain that they would lead back to such a condition.

Alienists and medical experts should be careful not to give too much latitude to the plea of insanity in cases like these, unless epilepsy be clearly recognized and proved; otherwise the extraordinary and absurd verdict rendered in the case of General Cole, of New York, may be repeated,—namely, "*Sane one moment prior to the homicide, insane during the homicide, and sane one moment after.*" Such a plea in a criminal case based upon the questionable theory of "transitory insanity" (without epileptic complications) is a prostitution of medical expert testimony.

ALCOHOLIC INSANITY.

Under this designation we include "mental disorder from the use of alcohol in both the acute and chronic forms.

"Acute alcoholic mania may be caused by a single excess in drinking, which in some individuals is always attended with

maniacal symptoms. It may constitute the alcoholic trance described under the head of transitory insanity. From long drinking and exhaustion or by withdrawal of the accustomed stimulant we may have the familiar *mania-a-potu*, or delirium tremens." (Folsom.)

"To the prolonged use of alcohol, primary delusional insanity, melancholia, mania, and dementia are often due. From continued alcohol-drinking, even in but slight excess, during many years, it is rare that some mental impairment does not become evident, if only an 'uncontrollable violence of the instincts and emotions,' a sort of moral insanity. . . . The prognosis is more favorable than in most forms of insanity uncomplicated by the abuse of alcohol, especially in cases of primary delusional insanity, if the bad habits can be effectually corrected and if the alcoholic excesses have not been continued long enough to produce organic changes in the cerebral blood-vessels. In the latter case the dementia sometimes simulates that of general paralysis so closely as to be called pseudo-paralytic dementia from alcohol."

"*Chronic alcoholic insanity* depends upon the vascular and other changes due to abuse of alcohol so long continued that the pathological condition has become organic and incurable. It is commonly associated with delusions or suspicions of persecution. It may be a purely moral insanity, with gross beliefs rather than distinctly insane delusions, and it rarely fails to be at least that when the persistent excessive drinking is kept up until the age of beginning dissolution of the brain. It then gives rise to all sorts of embarrassing complications in regard to property, family relations, and wills. Chronic alcoholic insanity may take the form of mild dementia, by virtue of which the patient cannot control himself, but can be easily kept within bounds of reasonable conduct by various degrees of restraint, from the constant presence of a responsible person to the seclusion of an asylum. In well-marked cases this dementia is associated with muscular weakness, tremor, and exhilaration to such an extent as to simulate general paralysis. It is then called by some (especially the French) writers *pseudo-paralytic dementia* from alcohol. The condition is susceptible of improvement by removal of the cause, alcohol, and by a carefully-regulated life, hydropathic treatment, etc., but complete recoveries cannot be expected." (Folsom.)

INSANITY AND BRIGHT'S DISEASE.

Alice Bennett, in an article on "Insanity as a Symptom of Bright's Disease," says, "Briefly formulated, my experience has led me to believe: (1) That, contrary to the generally received opinion, affections of the kidney are very common among the insane. (2) That 'uræmic poisoning' is one of the most frequent causes of insanity. (3) That, while the mental manifestations may be as varied as there are different centres subjected to irritation by these unknown poisons, the most prominent and constant symptom is some form of *mental pain*, which may range from simple depression, through all degrees and varieties of delusions of persecution, self-condemnation, and apprehension, with or without hallucinations, up to a condition characterized by a frenzy of fear, with extraordinary motor excitement and rapid physical prostration,—the 'grave delirium' or 'typho-mania' of some authors. (4) That the motor centres are specially liable to affection, as evidenced by the restlessness and incessant activity of many cases, less frequently by convulsions and convulsive twitchings, occasionally by choreic movements, occasionally by cataleptoid states."

"I fear that Dr. Bennett will not be generally supported in such a confession of faith. We may venture to affirm that as many cases of kidney-disease will be found in the ranks of our usual patients as among the insane, excluding general paretics. I have made this point a subject of careful attention for some years, and I have not observed the proportion of kidney-lesions which Dr. Bennett appears to have done. In the few cases of 'grave delirium' which have come under my care this point has been especially watched, but with negative results, as likewise in the majority of instances of mental depression and anxiety.

"I recognize the merits of Dr. Bennett's contribution, not the least of which is the emphasis which she puts upon the fact, before an audience of general practitioners, that insanity is a symptom the explanation of which must be looked for in some disturbance of the general physiological functions."*

* Brush, Annual of the Universal Medical Sciences, Sajous, 1891.

"INSANITY FOLLOWING INFLUENZA.

"Recent epidemics of influenza have been followed by numerous cases of insanity, and a few cases have been narrated of recovery apparently hastened by an attack of the epidemic. Metz narrates a recovery from paranoia. Febrile delirium during an infectious disease is, in fact, an acute attack of insanity. There are the febrile mental derangements proper to the fever (*psychoses fébriles*), and there is the delirium of convalescence (*psychoses asthéniques*). The latter embraces many distinct kinds. Towards the end of acute infectious diseases there is the 'delirium of inanition,' which may go on to the delirium of collapse, so well described by Weber. But, though asthenic delirium is the most common kind during convalescence (Christian), other kinds are met with, sensorial delusions being often present. There is probably, in such cases, a cerebral intoxication due to microbic products of the virus which has set up the disease. One great distinction between the psychoses of convalescence and the delirium of fever lies in the evident influence of heredity and the personal antecedents of the patient upon the character of the delirium in the former case (Krapelin, Savage), in contrast to its uniform course in the latter; in fact, heredity appears to play the chief part, and the acute disease is often only the accidental cause of the mental alienation. The diseases most frequently followed by the latter are: acute rheumatism, pneumonia, small-pox, intermittent fever, typhoid fever, cholera, and erysipelas; more rarely angina and scarlet fever, and very rarely measles and whooping-cough. As to influenza, the psychoses which may follow it were scarcely ascertained before the last great pandemic. Pétrequin, who described the epidemic of 1837, only cites Rush, of Philadelphia, and Bonnet, of Bordeaux. The former says, 'several persons who were affected by it had symptoms of madness, one of whom destroyed himself by jumping out of a window.' Bonnet reports a case of furious mania after influenza during the epidemic. Crichton-Browne gives a case of acute dementia after the influenza of 1874.

"Revilliod, of Geneva, has studied the nervous forms of 'la grippe,' and has shown that delirium may be the first symptom, as in other acute diseases. . . .

"Krapelin gives two cases of typical delirium tremens after in-

fluenza, the author one. Medico-legal questions have arisen out of the mental derangements of convalescence from acute diseases. Murders and other criminal acts have been committed, especially after intermittent fever. Thus influenza, like other acute diseases, may be the last stroke in developing an attack of insanity the kind of which is altogether independent of influenza. Even general paralysis has followed it. Influenza in itself never causes insanity. [Brush goes on to explain that an enfeebled system, the influence of heredity, and the personal antecedents of the patient are correlated facts of great importance.]

"Lastly, *la nonna* is a grave nervous form of mental *sequelæ* of influenza. . . . At Zozzoi di Sorramontane ten persons are said to have died of *la nonna* within a few hours. Pagello and Murer, who officially examined into this matter, found that they died of a sort of 'exanthematous miliary contagious fever,'—a comprehensive term. They also declared that not one of the patients had died within a few hours: even the most rapid cases had lasted at least two days. The Vienna physicians consider *la nonna* as an asthenic psychosis, ending in lethargy and coma, occurring, for the most part, in persons overworked and exhausted in every way, who have not been able to attend to themselves during the influenza. The Italian word *nonna*, literally *grandmother*, may also be translated *old woman* or *sorceress*. Now, there is a well-known legend that when a sorceress touches a sick person with the end of the finger he will surely die. The power of auto-suggestion is now sufficiently known, and, in a superstitious country, such beliefs must occasionally have sad consequences, especially among convalescents from a depressing disease like influenza."*

"The prognosis of all these psychoses is usually favorable, and the treatment is self-evident: it should be strengthening in every sense of the term, and also soothing."

INSANITY COMPLICATING HEART-DISEASE.

As to the alleged fact that *valvular disease of the heart* is regarded by some writers as a frequent cause of depressed emotional conditions, I fully agree with Spitzka, who states, "When, however, we remember the large number of persons whose hearts are in the

* Brush, Annual of the Universal Medical Sciences, Sajous, 1891.

most extreme conditions of organic failure, and who die in consequence, but without having manifested any special psychical disorder, we will, when we discover a fixed delusion of persecution in a subject with aortic obstruction, look for some other cause, such as an insane predisposition or mental overstrain, as the primary determining element, while the cardiac disorder may be admitted to act as an exciting cause, or, more accurately speaking, to determine the anxious or suspicious character of a delusion. It is a fact that patients suffering from cardiac lesions are more likely to develop anxious and suspicious delusions than those of an opposite nature."

Spitzka quotes Emminghaus, as follows: "In two cases of Basedow's disease (exophthalmic goitre) he found pronounced mental disturbance in the shape of melancholia and periodical mania. The occasional occurrence of this disorder in members of families afflicted with a morbid heredity would seem to indicate that the physical disease and the insanity are simply collaterals, and that both are the expressions of the same fundamental neurotic vice. It is an interesting problem for the future to solve why enlargement of the thyroid gland should in two disorders such as exophthalmic goitre and cretinism be associated with mental disorder or defect."

In answer to Spitzka, I would suggest that Schroeder van der Kolk, to my mind, very satisfactorily solves this problem as follows: "Therefore, without prejudice to other functions which are possibly also intrusted to it, the thyroid may be regarded as a diverticulum or reservoir by which a too strong pressure of blood may be diverted from the brain. Moreover, the position of the origin of the thyroidæ, in proximity to the vertebralis and carotis interna, yet gains in significance as this arrangement is not limited to the mammalia. For according to J. Simon (*On the Comparative Anatomy of the Thyroid Gland*, in *Phil. Transact.*, 1844, page 295), the thyroidæ of the bird arise exactly opposite the place where the carotid and vertebral come off, and even in amphibia and fishes these vessels stand in relation with the vessels of the brain.

"From this it becomes not improbable that the presence of a goitre, which receives a great deal of blood, and which may derive too powerfully from the brain, induces in cretins a weaker development of the brain, or, at least, a debilitated energy of it, although

cretinism is not to be regarded as a product of the goitre, but only appears to be frequently present with it. It might also in this way be explained why individuals who are afflicted with goitre for the most part are not very lively and active, but appear to be more phlegmatic. It may also be connected herewith that I have sometimes, in meningitis chronica and meningitis idiopathica, found a small indurated yellow-tinged thyroid gland, because through this condition the derivation of the blood-stream from the brain would be impeded, which then led to repeated congestions and contributed to the development of meningitis. I should at least wish this point to be regarded in future examinations."

LECTURE XVII.

EPILEPTIC INSANITY AND ITS MEDICO-LEGAL RELATIONS.

GENTLEMEN,—It is only of late years that epilepsy has received at the hands of writers upon forensic medicine the attention which its importance demands. In the criminal records of civilized countries but few cases will be found in which this common affection has been duly considered in fixing the responsibility of the accused, while in many instances but small appreciation has been shown of the grave questions involved in the study of epilepsy,—a disease of most changeful aspect and easily misunderstood.

The older cases of Tyler, Bethel, and Winnemore, discussed in Dr. Ray's great work upon "The Medical Jurisprudence of Insanity," bear witness to the fact of jurisconsult ignorance respecting this disease, even at a comparatively recent period ; while the later cases of Max Klingler and David Montgomery show how great has been the advance of a portion, at least, of the medical profession in knowledge of the affection, and, at the same time, the slow pace at which the legal follows the medical mind where questions of criminal responsibility are involved.

We concur fully in Spitzka's opinion, that when the medical or legal relations of epilepsy and epileptic insanity are discussed by authors, they confine their attention too exclusively to three categories,—“first, to the condition called epileptic mania ; secondly, to epileptic dementia ; and, thirdly, to the peculiar change of character which many epileptics manifest.”

Spitzka particularly insists that these phases fail to include many important conditions allied to epilepsy and dependent upon it, and which, moreover, may require special medical treatment and demand the serious attention of every thorough and conscientious medical jurist.

This author also remarks that it is an opinion quite prevalent with many, that, except for the period just preceding and following

the attack, and included in it, an epileptic, if not chronically demented, is always sane from a medical, and competent and responsible from a medico-legal, aspect. This view, he asserts, is held by many general practitioners and by most English medico-legal writers. On the other hand, he observes that certain examiners, as soon as they determine the slightest indications of epilepsy, instantly conclude that the subject cannot be of sound mind or responsible for any action whatsoever. This view he regards as peculiar to those who are frequently called in by the defence, where a plea of insanity is the last resort of the defendant. Both views, he states, constitute utterly erroneous extremes, and not erroneous only, but also damaging to the cause of justice, inasmuch as interested or even unscrupulous medical witnesses have been able to fortify themselves by such opinions drawn from published works, in support of testimony too often successful in defeating the legitimate purposes of the law.

I believe that most writers on this subject are prone to adopt extreme views. "*Virtus stat in medio.*" In the investigation of disputed scientific problems, especially those of a disease so occult, intricate, and variable as epilepsy, a wise conservatism is a more judicious and a safer guide.

We can scarcely in this matter go so far as to affirm with J. B. Friedreich, of Bavaria, a noted German authority, that "*Criminal responsibility is absent in epileptics, even should it be proved that the determination to commit a criminal action resulted from revenge or malignity.*"

On the contrary, I believe that Trousseau is nearer the truth when, in speaking of the criminality of epileptics, he uses the following strong language: "*It may be said, almost without fear of making a mistake, that if a man suddenly commits murder, without any previous intellectual disturbance,—without having, up to that time, shown any symptoms of insanity, and if not under the influence of passion, or of alcohol, or of any other poisonous substance which acts with energy on the nervous system,—it may be said, I repeat, that the man is afflicted with epilepsy, and that he has had a fit, or, more usually, an attack of epileptic vertigo.*"

Again, says the same author, "Who can calculate the degree of liberty possessed by a man in this state of transition between the actual attack and the complete recovery of his mental faculties?"

Is there a medical man bold enough to pronounce on this point, and to affirm that a crime committed after the attack must entail responsibility?"

According to Esquirol, the return of reason after epileptic seizures may sometimes manifest itself immediately after an attack, while in other cases it does not appear for several hours, or even days. Hallucinations, more or less permanent, may *complicate* epilepsy, and thereby become a source of dangerous impulses. It is known that when under rare conditions a threatened attack of epilepsy has been averted, its suppression in some instances occasions such painful and insupportable agitation as to cause some of the unfortunate victims of this affection to indulge excessively in alcoholic liquors, or to seek an occasion of quarrel by means of which to give vent to their pent-up irritability, and thereby to relieve excessive nervous tension, as an impatient hysterical woman does when she screams, stamps her foot, or falls into convulsions. Esquirol, when classifying the epileptic manifestations of three hundred and thirty-nine patients thus afflicted, who were under his care, speaks of some who have "un délire fugace," and again "soixante n'ont aucune aberration de l'intelligence, mais elles sont d'une très-grande susceptibilité, irascibles, entêtées, difficiles à vivre, capricieuses, bizarres; toutes ont quelque chose de singulier dans le caractère." Epileptic vertigo is more destructive to the intellect than convulsive seizures. Its duration is often inappreciable, and so slight, occasionally, are its manifestations, that persons—even medical men—who are not expert may readily fail to recognize it,—a fact of great importance, as we shall find further on. In describing the desperate and dangerous seizures of epileptic fury, Esquirol observes, "Cette fureur est si redoutable et si redoutée que j'ai vu un hospice du Midi où tous les épileptiques étaient enchaînés chaque soir sur leur lit, par la crainte qu'ils inspiraient."

It is much to be regretted that a fashion prevails at the present day of attempting to shield *criminals* under the protective ægis of a plea of insanity. This plea is too often set up to defeat the ends of justice and in extenuation of most fearful crimes. Insanity is a well-recognized cause of impulsive crime, and a suspicion of its existence should incite great care and the most elaborate research in dubious cases, but the fact that such a possibility of insanity is too frequently urged should not so affect public opinion

as to render punishment inevitable to fellow-creatures unfortunate enough to be morally irresponsible. Christian charity, or even common humanity, should make us at least fair and just in our dealings with this class of criminals, whose dark and frightful deeds are only results of a paralysis of that will-power which alone makes man a free and responsible agent. Less than this philanthropy cannot ask, and it were better that many guilty should escape than that one innocent man should unwarrantably suffer the extreme penalty of the law.

The disastrous influence of epilepsy upon the intellectual faculties is denied by the eminent author J. Russell Reynolds, as follows: "A prevalent belief is that some form or degree of mental deterioration is necessarily associated with epilepsy. The result of inquiry upon this point is to show that there is no such 'necessary' relation. The general belief is, however, to be accounted for partly by the strong impression which some notable cases of mental failure have made upon the minds of those who witnessed and recorded them,—such strong impression being followed by an undue inference,—and partly by the fact that the words '*epilepsy*' and '*epileptic*' have been made to include every form of disease of brain, spinal cord, or other organs which might be associated with fits, and also every variety of that multiform derangement which we call 'insanity of mind.' It is desirable, again, to assert that this article refers only to such cases as constitute epilepsy proper, and that the statistics upon which my results are based can only with a double injustice be compared with those derivable from lunatic asylums. A patient may be epileptic and a lunatic; he may be epileptic and an asthmatic; but there are some epileptics whose minds are as healthy as their lungs; and, so far as the natural history of epilepsy generally is concerned, it is a mistake to derive it from complicated cases." *

Notwithstanding these ingenious statements, I must tell you that the fatal influence of epilepsy upon the intellectual faculties, and especially of *epileptic vertigo*, is only too familiar to attentive and unprejudiced observers.

In many individuals of apparently perfect intellectual activity and organization, "a singular changeableness of feeling, of temper,

* Reynolds, article "Epilepsy," System of Medicine, vol. ii., 1868.

and of character, violent fits of passion, which they cannot master, point to a particular mental condition which in the greater number of cases will be followed by physical phenomena of a more distinct character, but always of the same order, as well as by more serious cerebral disorders, such as attacks of delirium, sometimes transient, sometimes *prolonged*, and then specially deserving the name of epileptic insanity. These disorders of an intellectual character may occur in the intervals between the epileptic paroxysms, may occur immediately before or after the attack, or they may be more or less prolonged, connected with, or occurring independently of, the attack, and are then more particularly characteristic of epileptic insanity." Such individuals are unquestionably subject, during the *interval between convulsive attacks*, to "a particular mental phase," quite significant as to certain tendencies and consequences. These patients are sometimes querulous and prone to acts of violence or explosive manifestations of the most terrific rage and sanguinary fury. Trousseau, in his admirable "Study of Epilepsy," quoting Jules Falret, says, "This irregularity in the state of their feelings and the degree of their intelligence is necessarily reflected in their talk and in their acts. Hence the excessive variability of their behavior towards those about them. For a certain period of their lives they are laborious, punctual, attentive to the duties of their profession, obedient and docile, and those who live with them, or who employ them, find their intercourse agreeable, or are pleased with their services. But at other times their conduct becomes *suddenly* modified, and presents the greatest irregularities. They are then incapable of fulfilling their duties, become negligent, lazy, and indolent. They forget the most elementary things, waste their time or wander here and there without aim or object in view, and are themselves conscious of the vagueness and confusion of their ideas. The most deplorable tendencies and the worst inclinations develop themselves in them at the same time; they become liars and thieves; *they pick quarrels with those around them, complain of everything and of everybody, are very easily irritated for the slightest cause, and even frequently commit sudden acts of violence*, which in most cases have not the excuse of provocation on the part of the victims of these acts."

The existence of "epileptic delirium," a condition somewhat allied to somnambulism, where the patient is not always totally

unconscious, as he is during the more common epileptic seizures, but has a vague and dreamy sense of his condition and of passing events, is fully recognized by our most reliable authors. The perceptive faculties may be unaffected, but the higher intellectual faculties and the will are not exerted, or are so only automatically. The actions of the patient are to a great extent instinctive, or in some instances purely automatic. These attacks hold "an intermediate place between simple epileptic vertigo and the convulsive fits." It is not difficult to appreciate the existence of instinctive actions, or even of actions springing from a still more subordinated source, or indeed of a purely perceptive origin. Ideas are purely mental operations, and must be held to originate in the cortical structure of the cerebral convolutions. That "mere sensation and apparent volition may exist independently of intellectual action, and even after the cerebrum has been destroyed," is an allegation of physiological psychology. Apparent volition without higher and more developed intellectual elaboration, by which an act is accomplished without the obvious concurrence and approval of the judgment, and without the co-operation of an active intelligence, appears to exert its influence by and through the gray substance of the hemispheres, and may be allied to the process styled "unconscious cerebration;" while, on the other hand, actions which are perfectly free, deliberate, and responsible are conceived, scrutinized, determined upon, and recorded, in the ganglionic cells of the gray matter of the hemispheres, before the will-force, which is determinative and free, issues its mandate, through appropriate and correlated regions of the motor zone, to the subservient voluntary muscles.

Thought, however, does not necessarily lead to expression or the induction of psycho-motor action in all cases. As M. Allen Starr appropriately observes, "Expression may be restrained; the impulse may be arrested. This restraint of the flow of thought outward in expression has been termed inhibition; and inhibition, or the act of control, is the highest of all cortical functions. . . ."

"In this summary of cortical action, it will be noticed that no mention has been made of the higher mental acts,—of judgment, reason, imagination, or of those qualities which determine talent and character. These cannot, as yet, be assigned to any particular region of the cortex, and no physical basis, no mechanism for such

purely mental acts, can as yet be pictured to the mind. As Hughlings Jackson has well said, 'Psychical states are not functions of any centre, but are simply concomitant with functioning of the most complex nervous arrangements.' It is certainly true that all mental activity has a physical basis in the brain; but there are numerous problems regarding the mutual relation of thought and cerebral action upon which physiology has thrown no light."

In evidence of the important fact that "the disturbance of the reason which follows a convulsive fit, and especially an attack of vertigo, is not always recognized so easily as it might be supposed," Trousseau cites the fact that "a medical man, for instance, is sent for to see an epileptic immediately after an attack. The patient answers questions pretty well to the point, follows out the doctor's prescriptions, and describes his feelings pretty accurately; but a few hours later has not only forgotten what occurred during the attack, as the rule is, but he has forgotten all the above circumstances, in which he had apparently concurred with so much presence of mind. It must, therefore, be concluded that his intellect had been deeply perturbed." "Not only," says the same author, "may the patient's reason remain in a perturbed condition for some time after the attack, *although a superficial observer may not perceive it*, but it sometimes happens that during the attack the epileptic seems to retain enough reason *to appear free*." How difficult, therefore, must it be to fathom the criminality of epileptics, if authority like Trousseau's, endorsed as it is by that of many distinguished psychologists, teaches us that inferences deducible from the uncontrollable impulses of epileptics demand such searching and careful study and watchful circumspection on the part of medical experts! Speaking of a patient, a magistrate and a very intelligent gentleman, subject to epileptic vertigo, Trousseau states, "He belonged to a literary society which held its meetings at the Hôtel-de-Ville de Paris. At one of these, during a discussion on an important historical point, he is seized with (epileptic) vertigo. He runs quickly down to the Place de l'Hôtel-de-Ville, and walks about for a few minutes on the quays, avoiding with success both carriages and the passers-by. On recovering himself he perceives that he has come out without his great-coat and his hat, returns to the meeting, and resumes with a perfectly lucid mind the historical discussion in which he had

already taken a very active part. He retained no recollection whatever of what occurred between the beginning of the attack and the moment he recovered himself. Now, had this patient quarrelled with and killed a man in the street, would a magistrate have believed that an individual who five minutes before and five minutes after was remarkably intelligent, and who during this pretended nervous seizure seemed to have his free will, could commit murder under the influence of an irresistible impulse? Sudden and irresistible impulses are of usual occurrence after an attack of *petit mal*, and pretty frequent after a regular convulsive fit. Patients should not be held responsible for their acts, whether these be followed or not by grave and painful consequences, the gravity of the act itself having nothing to do with the question. The individual is not a free agent for the time, and is, therefore, free from guilt."

The sudden outbursts of epileptic fury are so fearful and sometimes so disastrous that no maniac evinces greater or more uncontrollable passion. An individual so affected is to be dreaded by all around him, and may even become his own enemy. His violence is "blind and instinctive." The most terrible and *motiveless* crimes are perpetrated by epileptics during such an access of dangerous delirium, which may last from a few hours to twelve or fifteen days. It must be borne in mind that these fits of temporary insanity may sometimes be quite independent of epileptic seizures proper ("psychical equivalents"). Victims of the epileptic influence are apt to be irritated by everything about them, are inclined to wander in the streets, and to manifest a tendency to obey some concealed, quasi-mysterious influence, which irresistibly impels them to acts of violence. They are intensely unhappy, consider themselves persecuted victims, and, as in other forms of insanity, conceive a peculiar aversion to their friends and relatives, by whom they believe themselves especially persecuted. "If they have previously harbored any feelings of hatred or thoughts of revenge against any one, these feelings are quickened by their complaint, and suddenly roused to a pitch of intensity which prompts them to immediate action."

Epileptic insanity is essentially *impulsive* and instinctive. It is well known that epilepsy soon brings out in bold relief the animal traits of character, whose development seems to keep pace

with a slow but generally certain impairment of the intellectual faculties. Trousseau says, "The circumstance that repeated blows are struck and several wounds inflicted, or *several persons injured*, deserves to be specially noticed, and seems to characterize the condition of *furor epilepticus*. Hence it may be of considerable importance in a medico-legal point of view." I would particularly invite attention to the fact that after epilepsy has long subsided and is apparently cured it often breaks out in all its pristine intensity, a statement which is also true of the delirious form of the disease.

Hammond, in commenting upon the possibilities of "*irregular or abortive paroxysms*" of epilepsy, cites a very interesting and remarkable case which occurred in his practice during the autumn of 1875. "The patient, who was engaged in active business as a manufacturer, left his office at about nine A.M., saying he was going to a florist's to purchase some bulbs. He remained absent eight days. He was tracked all over the city, but the detectives and friends were always an hour or more behind him. It was ascertained that he had been to theatres, to hotels, where he slept, to shops where he had made purchases, and that he had made a journey of a hundred miles from New York, and, losing his ticket and not being able to give a satisfactory account of himself, was put off the train at a way-station. He had then returned to New York, passed the night at a hotel, and on the eighth day, at about ten o'clock, made his appearance at his office. He had no recollection of any one event which had taken place after leaving his place of business, eight days previously, till he awoke on the morning after his return to the city, and found himself in a hotel at which he was a stranger. It was ascertained beyond question that in all this time his actions had been entirely correct to all appearance, that his speech was coherent, and that he had acted entirely in all respects as any man in the full possession of his mental faculties would have acted. He had drunk nothing but a glass of ale, which he took with some oysters at a restaurant in Sixth Avenue."

This case is of vital importance in the medico-legal literature of epilepsy; its points are so salient that comment is unnecessary.

Eighteen years ago a case very similar to Dr. Hammond's fell under my care, in the person of a young lady from Louisiana,

affected with convulsive epilepsy. During the *post-epileptic stage* her actions were purely automatic for eight or ten days. She would execute the finest needle-work, read books and newspapers, or even converse with visitors, and yet remain utterly unconscious of all details pertaining to such actions. Suddenly returning to her normal condition, she recollected nothing that had taken place during the above-named period.

Hammond asserts that "Most, if not all, of the cases of '*double consciousness*' that have been reported are doubtless epileptic in character. An interesting case of the kind has been related by Azam. It is that of a young woman who, after having suffered from hysteria and convulsions, had two distinct phases of existence, living, in fact, two separate and different lives, and exhibiting different likes and dislikes and mental characteristics." *

Ray, in speaking of epilepsy and its legal consequences, in his "Medical Jurisprudence of Insanity," observes, "Another direct though temporary effect of the epileptic fit is to leave the mind in a morbidly irritable condition, in which the *slightest provocation* will derange it entirely. Sometimes this irritability is accompanied by a sense of anxiety, distrust, jealousy, and unfounded fear, and sometimes by great activity of the lower propensities. . . . Epilepsy seldom continues for any length of time without destroying the natural soundness of the intellect, rendering the patient listless, fretful, indisposed, and unable to think for himself, yielding, without any will of his own, to every outward influence, and finally sinking into hopeless fatuity or becoming incurably maniacal."

Again, from a medico-legal point of view, we find the following pertinent remarks of the same author: "To determine exactly the mental condition of an epileptic at the moment of his committing a criminal act is oftentimes a difficult task. It may have taken place in the absence of any observer, in a fit of fury *that rapidly passed away*, and which, perhaps, *may not have followed any previous paroxysm*; or the accused, though subject to the disease, *may not have recently suffered an attack*, and may have appeared perfectly rational to those around him. . . . Cases of this kind should be closely scrutinized, and where *the accused has been undeniably subject to epilepsy*, he should have the benefit of every

* Hammond, Diseases of the Nervous System.

reasonable doubt that may arise respecting his sanity. Less than this common humanity could not ask ; more, even, has sometimes been granted under the operation of milder codes than the English common law."

A great difficulty in the way of the scientific alienist is the prejudiced misconception, so commonly entertained, that no single faculty of the mind can become incapable of exercising its appropriate function without a necessary involvement of every other faculty. Yet the reverse of this proposition is probably true. The perceptive, emotional, and intellectual faculties may retain an apparent integrity, or even exhibit an unusual degree of vigor, while the volitional centres may be much at fault. On the other hand, the will may be normal, while one or all of the above faculties may be enfeebled. Indeed, I imagine that any one of these faculties, though they are so intimately correlated, may be exercised independently of the others.

Hammond furnishes the following forcible elucidation of the point under consideration : "(1) The brain may be so disordered that insanity is manifested only as regards the will. There are no false conceptions of the intellect, and no emotional disturbance, but solely an inability to exert the full will-power either affirmatively or negatively. (2) Many instances of 'morbid impulse' are uncomplicated cases of volitional insanity, in which an idea, suddenly flashing across the mind, is immediately carried out by the individual, although his intellect and his emotions are strongly exerted against it. Thus, a person who previously has not exhibited any very obvious symptoms of mental derangement—though careful inquiry will invariably show that slight evidences of cerebral disease have been present for some days—instantaneously feels a morbid impulse to commit a murder or perpetrate some other criminal act."

How careful, therefore, should we be in our deductions as to the responsibility of epileptic criminals ! Their disease is a constant source of irritation to a nervous system long since exhausted by continuous shocks, or "cortical explosions ;" hence a conspicuous morbid irritability,—"irritable weakness." The nervous system, like a Leyden jar, goes on accumulating its dangerous forces, to be finally expended in explosions of long-pent-up and concentrated violence.

I acknowledge that we are ignorant of the true pathology of epilepsy, and admit that this disease at times exhibits obscure and contradictory features difficult to explain, and which do not exactly correspond with the ordinary manifestations of epilepsy as generally described; but does it not seem presumptuous to maintain that criminality is therefore present, and that the prisoner at the bar should not have the benefit of the doubt? Does not the physician in his daily routine find cases of fever or of inflammation apparently similar, but varying so decidedly in type and in their special manifestations as often to test his diagnostic skill to the utmost? Do the maladies that we are constantly called upon to treat correspond faithfully with the typical and classical descriptions of the text-books? Can the study of any single case of a given disease thoroughly enlighten us as to the symptomatology and prognosis, or demonstrate to us the most efficacious treatment of the disease in its varying forms? If our experience as practitioners of medicine proves to us the folly of such a hope, should an unfortunate epileptic forfeit his life because the symptoms of his *disease* do not precisely correspond with "*hypothetical cases*" with which over-zealous prosecuting attorneys have burdened their memories after a night's cramming from standard authorities? Are the intricacies of mental disease susceptible of perfect elucidation by legal acumen? Must an unhappy epileptic receive a felon's doom under the weight of assertions declaimed before a jury, mostly untenable and unscientific?

Before proceeding further it may be well to consider the true meaning of the term "*responsibility*," which occurs so often with us. Dr. Bucknill's conclusions express my own ideas upon the subject, and are as follows: "Responsibility depends upon power, not upon knowledge, still less upon feeling. A man is responsible to do that which he can do, not that which he feels or knows it right to do. If a man is reduced under thralldom to passion by disease of the brain, he loses moral freedom and responsibility, although his knowledge of right and wrong may remain intact."

It appears, therefore, that an extreme difficulty exists in determining the criminal responsibility of epileptics, and that their mental condition is entitled to the most careful study, and should be regarded with the most elaborate circumspection by medical jurists.

Facts cited by such eminent men in science as Baillarger, Boileau de Castelnau, Delasiauve, Echeverria, Gray, Esquirol, Falret, Legrand du Saulle, Ray, Schroeder van der Kolk, Maudsley, and Spitzka go to support the same opinion.

The hallucination is generally forgotten after the seizure has passed away, but the manner, attitude, gestures, and acts are evidently referable to the fear or horror engendered by some imaginary danger,—imaginary, yet to its subject a fearful reality.

Epileptic delusions are often of a strong religious character; and these at first sight apparently harmless ideas may be the source of most sanguinary actions. They are usually of a homicidal character, although self-mutilation or suicide may be the form of culmination.

It is well known that epilepsy soon brings into bold relief the animal traits of character, whose development seems to keep pace with the slow but generally certain impairment of the intellectual faculties. Epileptics are commonly inveterate masturbators, and the crime of rape or sodomy may often be traced to the salacious tendencies developed under the brutalizing effects of the disease. Of course, where epilepsy of the masked form is pleaded in extenuation of crime of this sort, you should closely investigate the history of the accused for some evidence of epilepsy in some other form; examine him carefully for vices of conformation, which quite commonly accompany this formidable affection; test his memory, not only in a general way, but also particularly in reference to the acts which have brought him under the eye of the law.

An essential characteristic of criminal acts perpetrated by epileptics while under the influence of the special morbid psychical condition is their instantaneousness, their abruptness and suddenness. In two cases reported by Dr. Auzouy, where larvated (mental or cerebral) epilepsy was pleaded in extenuation of the crime of sodomy, that learned physician was enabled to determine the fact that epilepsy was not present by ascertaining the absence of these evidences of the disease, together with a perfect recollection on the part of the prisoners of all the incidents connected with the perpetration of the crime.

There is ample authority for the statement that total abolition of consciousness in these cases, although usual, is not at all neces-

sary, and that a state bearing some resemblance to somnambulism may exist. Dr. M. G. Echeverria has clearly elucidated this subject in an article of very high value in the *American Journal of Insanity* for January, 1873. He shows by citations from the works of Delasiauve, Legrand du Saulle, Boileau de Castelnau, and Trousseau, and from cases in his own practice, which has been remarkably rich in epilepsy, that premeditation and action upon such motives as revenge, jealousy, etc., are by no means uncommon in epilepsy.

Recognizing, therefore, a frame of mind to which epileptics are obnoxious, which is strange, indescribable, and *sui generis*, the result of a morbid condition over which they have no control, and for which, therefore, they are not responsible, how can we attempt to designate with precision the dubious confines where responsibility ends and criminality begins?

In relation to masked epilepsy, Maudsley, in his "Physiology and Pathology of the Mind," makes these observations:

"In such cases there are often sudden and vivid temporary hallucinations. Again, the mental disorder which sometimes takes the place of an epileptic attack, representing, in fact, a *masked* epilepsy, may appear as simple impulsive insanity. . . . It happens sometimes that the patient succeeds in controlling the morbid idea for a time, calls up other ideas to counteract it, warns his probable victim to get out of his way, or begs earnestly to be himself put under some restraint; but at last, perhaps from a further deterioration of nervous element through bodily disturbance, the morbid idea acquires a fatal predominance; the tension of it becomes excessive; it is no longer an *idea*, the relations of which the mind can contemplate, but a violent *impulse*, into which the mind is absorbed, and which irresistibly utters itself into action."

That the presumption of moral irresponsibility is in favor of the epileptic accused of crime may be fairly concluded from the weight of authority, from the facts I have given, and from a humane application of the rule that the accused is entitled to the benefit of any reasonable doubt of his responsibility and culpability.

The following remarkable passage by Ray (Trial of Winnemore, *American Journal of Insanity*, October, 1867) shows that this venerable Nestor of American psychology arrived at the same

conclusion nearly a quarter of a century ago: "In view of what we already know of epilepsy, and what still remains to be learned, we have a right to require the utmost circumspection, and the closest investigation, whenever the legal liabilities of epileptics are in question. The fact of its existence being established, is it going too far to say that legal responsibility is presumptively annulled, and that the burden of proof lies on the party that alleges the contrary? People are scarcely ready for it yet, perhaps, but to that complexion will they come at last."

In cases, also, in which heredity and the neuropathic temperament exist in combination with a history of cranial injury, or where symptomatic manifestations of focal lesions of the motor tract are indicated by "the *signal symptom*" of "*Jacksonian epilepsy*," the presumption of doubt in criminal cases must always be in favor of the defendant.

In this connection Forbes Winslow makes the following observations: "Do we estimate in a manner commensurate with its grave and vital importance the necessity of watching, with the most scrupulous care, the cerebral symptoms that follow all mechanical injuries to the head? I am satisfied that a vast amount of organic, chronic, and incurable disease of the brain and disorder of the mind can be directly traced to this cause. In many cases positive and undoubted evidences of disease of the brain are present without exciting a suspicion as to the cerebral origin of this affection, or character of the symptoms. A man receives a blow upon the head. He may suffer from partial concussion of the brain, or be merely stunned. He recovers without any apparent inconvenience from the injury, but subsequently head-symptoms exhibit themselves, clearly the consequence of the injury which the brain had sustained many years previously. I am satisfied that the importance of this subject cannot be exaggerated. Repeatedly have I had cases of epilepsy bidding defiance to all treatment, tumors, abscesses, cancer, softening of the brain, *as well as insanity in its most formidable types*, under my care, whose origin could unquestionably be traced back, for periods varying from eight to ten, fifteen, and even twenty years, to damage done to the delicate structure of the brain by injuries inflicted upon the head!"

Confirmatory of Forbes Winslow's views, Griesinger ("Mental

Pathology and Therapeutics") makes the following observations: "It frequently happens that on minute inquiry the physician learns from the relatives of the patient of former circumstances of this kind which had been almost forgotten,—a severe kick from a horse, a fall or blow on the head which was followed by insensibility. Sometimes the friend now remembers, for the first time, that since the accident a certain change had taken place in the character of the patient,—that he had become fretful, irritable, perverse, etc. This change, however, had been little heeded, and had not even been recognized in its true significance as a precursor of insanity when the disease broke out."

The following details of a recent case in my practice confirm these views. I have treated for the last three years a case of convulsive epilepsy, in one of the most intelligent merchants of this city. After an exhaustive analysis of the history of the case and most careful study of its etiology, my diagnosis was idiopathic epilepsy. This summer, while conversing with the patient, my attention was attracted to an ugly scar upon the scalp, in close relationship with the upper portion of the left ascending frontal convolution: its presence was revealed because the patient had had his hair very closely clipped on account of the extreme heat which then prevailed. I was much surprised at finding it, and inquired as to its cause: the patient replied that he had received a severe kick from a horse in that region when a boy, which had rendered him unconscious for several days, but that he had not thought the fact worth mentioning. Thus a serious omission had occurred in his recital, notwithstanding all my care, for it is my custom to investigate with the utmost detail the possibility of traumatism in all cases of disease of the nervous system, and especially in epilepsy and insanity. With intent, therefore, to avail himself of the resources of cranial surgery, in all appropriate cases of epilepsy and insanity at least, the physician should cause the scalp to be shaven, whether the patient admits any traumatic history or not. Previous injuries to the head, moreover, are frequently kept from the knowledge of young people by their parents, especially when nervous or mental symptoms supervene, so that in later years the patient may have no recollection of an injury, and thus be unable to furnish a most important hint to his medical examiner.

Regarding temporary hallucinations, to which epileptics are so

subject, Hammond observes, "We all at times momentarily have hallucinations and delusions, but the judgment at once prevents continued deception. When this fails to be the case, delusions exist, and we are the subjects of intellectual insanity."

Maudsley observes, "Sometimes an attack of mania notably precedes an epileptic fit, or a series of epileptic fits; but it is not so clearly understood that the mental derangement so occurring may have the form of profound *moral* disturbance, with homicidal propensity, but without manifest *intellectual* disturbance."

Another fact to be continually borne in mind by medical experts is the possibility of the existence of "*epileptic vertigo*" in the entire absence of convulsive attacks or any other epileptic complications. In this connection the affirmation of Schroeder van der Kolk should be always remembered, that epileptic vertigo "depresses the mental powers much more rapidly than spasms without loss of consciousness;" by this latter expression apparently intending to designate "Jacksonian epilepsy," which had not been strictly differentiated in his day.

It may be argued that the extenuation in these cases is far-fetched, and that the ingenuity of doctors and the evasive skill of lawyers are prejudicial to the interests of the community, whose sense of justice is daily outraged by the ever-ready plea of insanity. Such a conclusion is unfair and fallacious, and deserves but little consideration. Science is truth: because an abuse exists we are not called upon to abate our scientific zeal in cases which, though obscure and marvellous, are yet within the scope of scientific analysis. "If any one supposes that the marvellous is incompatible with true science, deserving only rebuke and derision, let him consider that every step in the progress of science has been but the repetition of a marvel, scouted at first as unworthy the serious attention of the philosopher, and welcomed at last with triumphant admiration and joy."

I would, moreover, repeat that in cases of alleged epilepsy the *apparent* absence of epileptic convulsions for years is no proof of the *cure* of the disease. The morbid basis may still exist as "masked epilepsy," epileptic vertigo, or nocturnal epilepsy, and these forms may linger, bidding defiance to ordinary investigation unless conducted by those who are on the watch for the many subtle forms of this extraordinary disease.

Epileptic dementia may terminate in stupor, imbecility, or idiocy (Esquirol), which, as Spitzka observes, "is intimately dependent on the frequency of the convulsive attacks, . . . according as these attacks begin later or earlier in life. Aside also from those attacks of furious madness or purposeless automatism replacing the convulsive attack, and which may be regarded as *psychical equivalents* of the convulsion, there are forms of more or less protracted insanity which follow some individual epileptic attack, or break out in the interval, or finally extend over the entire interval, which are to be strictly distinguished from these forms."

Spitzka quotes Samt as including both the "*petit mal* and *grand mal intellectuel*" of Falret under the head of acute post-epileptic insanity, and defines the latter as insanity immediately following the convulsive paroxysm, and pursuing an acute course. He subdivides this acute form into—

1st. *Simple post-epileptic stupor*, which may be complicated with dreamy delirium, etc.

2d. *Post-epileptic morbid conditions* of fear or fright, either simple or complicated with *délire raisonnant* or great excitement.

3d. *Post-epileptic maniacal moria*. This form is rare, and simulates ordinary acute mania to such an extent that even the expert may be deceived. Spitzka believes the treacherous and malicious character of the violence will enable us to distinguish this disorder from ordinary attacks of acute mania.

Under the head of *chronic protracted epileptic insanity* Spitzka says Samt describes many cases which are evidently related to the post-epileptic forms.

Spitzka adds, "Just as the forms characterized in Samt's classification were designated post-epileptic, these latter, which are far from infrequent, deserve to be designated as *prodromal* or *pre-epileptic*. If the chronological relation of the mental disturbance be made a principle of classification, much confusion could be avoided by adopting the following order:

"1. *The epileptic psychical equivalent*, which replaces the convulsive attack.

"2. *The acute post-epileptic insanity*, which almost immediately follows the convulsive attack. . . .

"3. *The pre-epileptic insanity*, which precedes the outbreak

of a convulsive attack or its equivalent, and increases up to the moment when the paroxysm explodes.

"4. The purely *intervallary epileptic insanity*, which, neither immediately following nor preceding a paroxysm, occurs in the interval between such."

Spitzka finally states that it is possible for all these forms to occur together, and in addition there is very apt to be a background of protracted epileptic dementia to complicate the picture.

In conclusion, with regard to the disposal of cases of this character,—as of persons acquitted of crime on the ground of insanity, whether of epileptic origin or not,—while I would extenuate their faults and seek to measure a full allowance of justice and of mercy to them, I would not forget what is due to the community at large. The homicidal lunatic should not be turned loose upon an outraged community, which must watch over its own preservation and enforce law, which alone can protect life and property and preserve the liberties of individuals. On the contrary, when an epileptic criminal's life has been saved by a plea of insanity, the sentence should be confinement in an insane asylum for life, where his dangerous and destructive propensities can be held in check and the public secured from alarm and injury. It matters not that he may be sane when acquitted, or that a sane man should not be incarcerated in a lunatic asylum: "*aux grands maux les grands remèdes*." No person who has been proved liable to explosive fits of homicidal insanity should be allowed his liberty during an apparent convalescence whose continuance no expert, however great his attainments or experience, can guarantee. Nor, on the other hand, is it just or humane that after acquittal for a homicide actually committed, such an epileptic should be submitted to the risks of another prosecution, for he might repeat the act and be sentenced to death at some subsequent time.

LECTURE XVIII

GENERAL PARALYSIS OF THE INSANE.

History—Etiology—Symptomatology—Diagnosis—Prognosis—Pathological Anatomy—Treatment—Cerebral Surgery in General Paralysis.

GENTLEMEN,—This formidable malady is one of the most destructive both to mind and body in the catalogue of diseases. I have never known a patient afflicted with it to be in the slightest degree benefited by treatment: its correct diagnosis is really his death-knell.

Medical literature has been flooded with able contributions to the study of this disorder.

Blandford justly credits French physicians with having first recognized and described it as a special form of insanity; but "the credit," he says, "must be divided among several." Esquirol was undoubtedly the first who held that insanity complicated with paralysis was incurable, but he did not perceive that the combination of these two elements essentially constituted the disease, regarding the paralysis as a complication of the insanity.

Bayle in 1822 maintained that as a sequence of chronic inflammation of the arachnoid a form of insanity was developed, and that "the mental disturbance and paralysis were synchronous." Delaye in 1824 wrote that the disease described above was a softening or atrophy of the brain, and was not always accompanied by insanity. Blandford further states that Calmeil in 1826 "gave a most complete account of it, and to him frequently is ascribed the merit of having been the discoverer." In 1859 he published a treatise on the subject, in which he set forth his latest views of its nature. All authors who have contributed to the literature of insanity since then have devoted much time and labor to the investigation of this subject.

"Of the many divisions of general paralysis into several clinical types, all of them naturally more or less arbitrary, I know no other

so satisfactory as Meynert's eight. ('Klinische Vorlesungen über Psychiatrie,' Wien, 1890, Braumüller.)

"1. Simple progressive dementia, with the usual progressive motor impairment which accompanies it.

"2. With delusions of grandeur and with marked motor disturbances, which appear simultaneously and are progressive. The mental state is usually of exaltation, but there may be depression.

"3. Of the same type as the last, but lacking its steadily progressive character,—that is, with remissions.

"4. Cases in which the characteristic exaltation and grand delusions reach such an astounding height that manifest motor symptoms are looked for with confidence from day to day, and yet may not appear even for a year, any slight incoördination naturally being obscured by the general muscular disturbance. Meanwhile there may be such an improvement as to simulate a recovery.

"5. A very rare form, with alternate symptoms of exaltation and depression.

"6. With early furious delirium, painful hallucinations, confusion and incoherence somewhat resembling acute delirium.

"7. In which the characteristic indications appear secondary to other forms of insanity,—for instance, after paranoia or melancholia.

"8. The combined form, with sclerosis in the whole cerebro-spinal tract, the symptoms of tabes or spastic paralysis predominating according as the posterior or lateral columns of the spinal cord are chiefly involved. The ascending type, in which the cord is first affected, is rare. Optic neuritis, ending in atrophy and paralysis, especially of the ocular muscles, may precede marked mental symptoms." *

Folsom, in his excellent monograph on "The Early Stage of General Paralysis," observes that "the impression of general paralysis, however, almost universal among officers of insane asylums and the medical profession, is of a disease in which the symptoms are early difficulty of speech, muscular tremor, and uncertain gait, accompanied by remarkable mental weakness, and

* Folsom, Some Points regarding General Paralysis, May, 1891.

in many cases *manie des grandeurs*." The only general medical text-books describing the obscure insidious stage are the second edition of Strümpell and Pepper's System of Medicine; and, except Meynert, the writers who have recognized it, so far as their opinions are given, agree with Voisin as to the impossibility of diagnosing general paralysis in this *early** stage.

"Indeed, the early period of this disease, as generally described, is a comparatively late stage, and, excepting a few writers, this statement holds good at least until a few years ago. With regard to the occasional articles in medical journals which have appeared from time to time, within the last forty years, on the prodromal symptoms of general paralysis, reference has for the most part been made to particular acts rather than to a general mental state."

"In this country, Spitzka, in his treatise on insanity, in 1883, defines the initial stage of general paralysis as so insidious in its development of symptoms that it is difficult to say anything positive as to its duration, and as marked by change of character, attributable to simple brain-failure, especially in negligence of ordinary duties, or indifference to them."

In this connection Folsom quotes Brierre de Boismont, who in 1860 published cases illustrating the initial stage of this disease, the most striking of which was that of a man who began thieving nearly eight years before the diagnosis of general paralysis was made. He then refers to Voisin, in 1879, and Luys, in 1881, who mention an obscure prodromal period, "with marked, although not distinctive, mental symptoms." Folsom then states that it was not until the appearance of Professor Ball's work in 1883 and that of Regis in 1885 that he found in French special medical literature a clear statement of the very insidious and obscure character of the *first* symptoms of general paralysis.

Folsom adds that since Sanders, in 1876, the leading German writers on diseases of the brain, especially Wernicke, Schüle, Krafft-Ebing, and Mendel, "have described the beginning of general paralysis as simply a change of character quietly developed, and, later, so slight an impairment of intelligence as to admit of patients continuing their usual occupations for a considerable time, without any indications of diminished mental power noticed,

* Italics my own.

except by those who come in contact with them most intimately, and by them not thought of as indicating disease."

"In Great Britain, Savage in 1884 alludes to a prodromal stage as attended with developed rather than initial symptoms; . . . and Sankey, writing the same year, fails to recognize so early a stage. Clouston, writing in 1883, does not describe the initial or prodromal stage. Mickle in 1886 mentions the prodromic stage, but with symptoms marking some progress in the disease, although he states that 'in the history of many a case do we find that some moral or other mental change in the patient, some perversion of the affective sentiments, has been noticed long before the acknowledged onset of the disease;' and in the mean time it has happened that fortunes have been wrecked or characters ruined.

"General paralysis usually begins so slowly and gradually that a definite period for its beginning can hardly ever be given. In addition, it is often clear, at a period when the disease is already fully developed, that certain early symptoms, whose nature was not at first correctly recognized, ought to have been regarded as the initial symptoms." (Strümpell.)

My own experience corresponds with that of the above-mentioned writers. In the very few cases that were under my observation *prior* to the development of the *general* disease, I have noticed several instances of an obscure prodromal stage.

In the case of a lady whom I have recently treated I had excellent opportunities of observing the correctness of this statement. I have been her family physician for over twenty years. She has been confined in an asylum for the last eighteen months, the diagnosis of her disease being *general paralysis of the insane*. For a year or two prior to her becoming an inmate of the asylum, while she was an admitted leader of society, she developed, although *previously above reproach* in her moral character, salacious propensities which were indistinguishable from erotomania, in consequence of which she was in perpetual trouble. I never saw a greater moral perversion and change developed without adequate ascertainable cause than in this particular case. Her intrigues became so notorious as to rivet public attention upon her. Her effrontery and importunities towards those of the opposite sex finally became so aggressive and disgraceful as in two instances to lead to her arrest. She spent money lavishly, and undertook

long journeys, from St. Louis to New York in one instance, and from St. Louis to Canada in another, for the gratification of her nefarious propensities. Yet during all this interval of time not the slightest mental defect could be ascertained, and she continued to control and to conduct most successfully an enormous estate. In every other respect she was seemingly an elegant, refined, and cultivated lady, nor could a most careful scrutiny determine the existence of symptoms of any stage of the *developed* disease.

It is customary to divide this disease into different periods or stages: the distinction, however, is to a certain extent an arbitrary one, because the features of each stage are not always characteristic, and either period may merge into the other.

Blandford makes the following division:

"1st. The commencement, or period of incubation.

"2d. The acute maniacal period.

"3d. The period of chronic mania lapsing into dementia, with utter prostration both of mind and body."

It is a matter of great regret that the earlier stages of this direful affection so frequently seen by the general practitioner often remain utterly unappreciated or even not recognized. Seguin most appropriately laments that, "even by neurologists, the diagnosis of nervous prostration or cerebral fatigue is often made and a delusive prognosis given. Rest and change are advised, when an active medication and seclusion from excitement should be prescribed."

The same author further states the symptoms by which an *early* diagnosis can be made, as follows:

"*A change in the patient's moral character,—ethical changes.* Ethical development is the last and highest phase of action or function of the cerebrum in mammals, and more strikingly in man; it is the least instinctive or organic function, a sort of delicate efflorescence; and, consequently, it is not surprising that it should be the first to retrograde when the cerebrum is undergoing wide-spread degeneration of slight degree. The alteration, allow me to repeat, is a positive *change*, not an accentuation to a morbid degree of the patient's previous faults of character, as is observed in various forms of insanity. Diminished regard for decorum, slovenly habits in dress and at table, slight deviations from truthfulness, an inclination to or relish for ribald or obscene jokes,

actual indecency in language and acts, indulgence in stimulants, lascivious familiarities and visits to houses of prostitution, etc., in a man who previously never lapsed in such matters, should always cause the greatest concern and lead to a suspicion of beginning diffused encephalitis.

"Irritability or abnormal anger might be included in this list, but this increased reaction to external stimuli is a symptom more characteristic of cerebral neurasthenia, hysteria, etc., and seldom means a change in character. Indeed, in my experience, good nature and abnormal pliability are more frequent than irritability in dementia. The same remarks apply to the abnormal emotions shown by victims of this disease: they laugh or cry 'hysterically' on the slightest provocation. This state, however, only means that we have an abnormally sensitive brain and diminished self-restraint before us,—conditions fully as frequent in simple cerebral neurasthenia as in dementia paralytica.

"Not rarely these symptoms though appearing very early are not known to the physician, because the patient cannot tell of them and his relatives are ashamed to reveal them. They must be sought for; consequently, although these ethical symptoms are of great importance, they cannot be designated as striking or as leading symptoms, except to the family physician, who, of course, has peculiar opportunities for noting these changes, even before the family is alarmed." (In a foot-note Seguin states, "I have sometimes first heard of these symptoms some time after the consultation, from associates and friends of the patient, the family having carefully concealed them.")

"Often it is believed by laymen and physicians that the alcoholic and sexual irregularities of the patient are causes of subsequent symptoms, but this is, most authorities agree, an erroneous and dangerous view. Doubtless minute changes in the brain precede the ethical degradation.

"Mental dulness and inaccuracy.—The patient often complains of these himself; he is becoming 'lazy'; mental exertion is onerous; he feels dull, and even drowsy, during business hours; he is conscious of doing everything slowly and laboriously. He notes mistakes in his calculations, failures to keep appointments, and other evidences of failing memory and impaired power of attention. In many cases—those in which a slight degree of exaltation

appears early—the patients are unaware of these faults and energetically deny them. Yet it is astonishing how long professional or old habitual acts continue to be performed with tolerable exactness, even after many symptoms have appeared. Such mental operations as have by long practice become almost automatic or semi-instinctive resist the disease remarkably.” (In a foot-note the author continues, “I have known of physicians who attended to ordinary practice fairly well (but were suspected of drinking by their clients) for a long time after the diagnosis was clear; and two years ago I made a diagnosis of dementia paralytica in a popular actor, who starred about the country with diminishing success for several months afterwards.”)

Folsom divides the symptoms of well-marked general paralysis into four distinct types: “the demented and paralytic, the hypochondriacal, with melancholia, with exaltation and mania.” He further adds, “there are mixed cases in which some or all of these forms occur.” As before stated, I prefer the simpler classification of Blandford, who divides the disease into three stages. I fully agree with Folsom that the demented form is the most common, and with Seguin that motor disturbances are often the first complained of, which, as observed by this author, consist principally “in difficulty and slowness of articulation, awkwardness of the fingers for delicate movements, impaired handwriting, visible tremor, a general loss of muscular force often perfectly evident to the patient in those cases where exaltation has not set in. I have notes of several cases in which the patient came to me spontaneously for difficult articulation (this appearing to him the only symptom).” Seguin further declares that “in very rare cases impaired articulation, and slight tremor of the hands, are really the first symptoms.”

Further on in his admirable description, he continues, “If there is a pathognomonic symptom in all semeiology, it is the peculiar faulty speech of the early stage of non-delirious dementia paralytica. It is as valuable for the diagnosis of this disease as fulgurating pains are for that of tabes. Yet a similar speech (or at least one that seems like it) is met with once in a while in a generally tremulous patient who has not cortical degeneration. So rare is this that I have record of but one example: so that the value of the symptom remains very great.”

In another place Seguin says, "As tremor underlies the faulty speech (or dysarthria) and the awkwardness, allow me to speak of it at some length. Different cases present tremors which I am in the habit of classifying as coarse and fine. Sometimes it requires the closest scrutiny to detect them, or they may be almost choreic in form. Always these tremors appear only on exertion; they are not, strictly speaking, fibrillary contractions, nor are they at all like the rhythmical or quasi-rhythmical movements of paralysis agitans, senile or alcoholic trembling. Consequently you will have to seek for this symptom by bidding the patient to do certain things. Sitting quietly, he appears free from tremors; bid him frown, and the muscles of the upper part of the face show tremulous action; bid him show his gums, the tremor appears in all the lower facial muscles; bid him protrude the tongue, and that organ appears filled with fine tremulous contractions, or is agitated as a whole by coarser tremors; make the patient hold out his hands, and various degrees of non-rhythmical tremor are apparent to your eye or to your fingers (if you hold his fingers within yours). Tell him to speak, and the aerial waves are broken by the irregular muscular contraction, producing various forms of faulty articulation. Emotion, which in almost all persons is accompanied by unconscious muscular movements in various parts, also brings out the tremors. At once when the patient begins to tell you his ailments or replies to your questions, the faulty speech reveals the tremors, and to the experienced ear foretells the man's doom."

Seguin lays *particular* stress upon the statement that the fundamental character of the speech in paresis is "non-rhythmic vibration," which leads to a jerky, irregular utterance, and an increased muscular tremor, causing the indistinct sounds with omission of syllables in long words. Seguin asserts that it is a mistake to regard this omission of syllables as *the* characteristic fault, as it is not present in the early period of the disease. The speech in paresis, he says, is not only tremulous or jerky, but often slow; some syllables are spoiled,—some are omitted from long words. No other disease causes this.

Starr, in his work on "Familiar Forms of Nervous Disease" (1891), says, "The onset of this disorder is protean in its manifestations. The earliest symptoms may be either physical or mental or both. Tremor of the fingers and of the hand in writing;

fibrillary tremor of the tongue and lips; slight difficulty in the pronunciation of certain words, such as occur in the phrase "Grief brings frightful dreams;" overaction of the occipito-frontalis muscle; slight irregularity or inequality of the pupils, myosis, or loss of the pupillary reflexes; exaggeration of the wrist-, elbow-, and knee-jerks (rarely diminution): all these are among the earliest somatic characteristics. Later these become more and more pronounced, until diagnosis is inevitable."

The temperature in general paralysis is higher than in health. It is rarely lower. Folsom reports that in the only extremely rapid cases he ever treated (two months in all) it was 97° F. or thereabout for a number of days, and then rapidly rose to 103° and 104°, where it remained almost until death. He very truly states that after the congestive, epileptiform, and apoplectiform attacks it rises from two to seven degrees, and remains high for a considerable time. Folsom divides the *leading* symptoms of general paralysis of the insane into (1) vaso-motor, (2) mental, and (3) physical.

This view is of great importance, if we admit Meynert's theory that preceding and causing the diffused cortical encephalitis there is a functional vaso-motor disorder which he considers curable.

According to Folsom, the vaso-motor symptoms are represented by arterial paralysis, changes in the cerebral circulation, diminished arterial tension, attacks of vertigo, dizziness, or faintness, confusion, incoherence, and dementia, elevation or depression of the temperature, frequent attacks of congestion or transient cerebral anæmia, emotional disturbances, epileptiform or apoplectiform seizures, with or without temporary loss of muscular power, monoplegic, hemiplegic, or paraplegic character, bed-sores, skin-affections, cyanosis, neuro-paralytic hyperæmia of the lungs, bladder, and intestines, cold feet, œdema of the skin and local sweatings, and variations in the mental state. As the disease advances there is an increase in the loss of power of control and in the loss of mental power, the two symptoms making progress side by side.

Folsom further states that "after the prodromal period has passed the mental impairment increases, so that the judgment, memory, power of attention, and expression grow progressively worse; and this impairment constitutes the only characteristic

mental state universally present in all stages of general paralysis of the insane,—namely, progressive dementia.” The impairment of the sense of right and wrong becomes quite marked ; the patient loses the sense of property and ownership. In no other disease could the reported case occur of a man, to outward appearance well, going up to a policeman and asking his assistance in rolling off a barrel of liquor which belonged to some one else, and which he meant to appropriate. For this reason, what seem to be thefts are very common, and, although by that time there is striking mental impairment, it may not be obvious to every-day people. Almost every other moral obliquity occurs, particularly a tendency to drunkenness and to every possible violation of the proprieties and laws regarding property and the sexual function.

Lastly, there are strange delusions, consisting of remarkable exaltations, a curious and overweening self-esteem. A proneness to extraordinary exaggeration and extravagant ideas is rarely absent. Indeed, it seems impossible for the patient to make the least statement without its being greatly exaggerated. These last peculiarities have caused the affection to be termed by the French *manie de grandeur*, or *manie d'exaltation*. Persons afflicted with this disease always entertain a conviction of their own importance and power, and invariably endeavor to impress this upon others with whom they associate. I will cite you a few instances which happened in my own experience.

I have now under my care a man who labors under the most remarkable self-exaltation. He imagines that he is the wealthiest man in the world, that he actually possesses all the gold in existence. With him money is of no value : he is immensely, incomparably rich, and states that if the waters of the Atlantic were withdrawn and its bed exposed, it could not contain all his gold. He imagines that all the civilized world recognizes in him the greatest living railroad king ; and, as he comes from Memphis, he considers that city the great centre of the globe. In its vicinity, he says, there are immense mountains of iron, which he purchased years ago. This enables him to build railroads in all directions, and he purposes to transport upon them all the larger cities of the United States to Memphis. He has now in course of construction, in Liverpool, immense derricks, by means of which he will raise the entire city of St. Louis into the air and place it upon a

line of railroad he is building for the purpose, which will be not merely an air-line road, but three miles, at least, above the surface of the earth. He is quite certain of being able to move our entire metropolis in less than thirty seconds. His resources are inexhaustible. His mountains of silver he claims to have received from God Almighty as an acknowledgment of the fact that he had lent Him a large sum of money upon some previous occasion. This man is as firmly convinced of the truth of these ludicrous ideas as you, gentlemen, are of your existence. His delusion is the more striking in view of the fact that he is very poor, and not even at liberty, but in an asylum.

An architect under my care some years ago had also a very remarkable idea of his own greatness. He was willing to extend favors to Almighty God, and had in contemplation the construction of a "temple for the world." He had made drawings which were really beautiful and complete in detail. He stated that to this temple all the people of the earth would come to bend the knee. It would be replete with gold, silver, and mosaic; there being, in fact, more of the former than all the nations of the world could supply, he himself having ample means of furnishing inexhaustible quantities. To show you the immense idea of grandeur which had taken possession of this man, he said that on the day of the inauguration of his temple the whole civilized and barbarian world would be present to celebrate the occasion. All of these would occupy the temple, together with all the illustrious dead of past ages; and yet the temple was not to cover more ground than the St. Louis County Court-house! The assembled multitudes would all come to worship the statue of the greatest of living men,—himself. His statue was to be of gold and on an eminence from which it could be seen by all the world. On Christmas-day and the Fourth of July he would mount the eminence in person, so that everybody might adore and venerate him.

We treated for many years a case of paralytic dementia in which the patient presented a very singular and interesting delusion. He was a very learned man, and had been for a long time a successful professor of chemistry. His body presented an unusually large crop of moles and warts, to each of which he attached a mythological or astronomical name. One he would call Mars,

a second Venus, a third Jupiter, etc. He had written in Latin and Greek upon the walls of his room many descriptions of the relationship which these growths bore to his sacred personality, constituting quite a long and learned history. By grouping them together and attaching thereto long algebraic formulæ, he proved, to his own satisfaction at least, that he was the God of Gods, the ruler of the universe. For years he never varied in the details of these descriptions, and it was quite interesting to listen to him. He was a splendid classical scholar, and, singular to say, notwithstanding the character of his disease, his memory in some respects was extraordinary. He could give long and most accurate recitations from Virgil, Horace, and Homer.

Such conceptions are peculiar and characteristic. One man may believe that he is the greatest of living generals; another, that he controls the universe, and that upon his care and attention the rotation of the earth depends.

It has been a matter of much discussion whether the symptoms of the disease succeed one another in a certain regular order,—whether the paralysis of the organs of speech, or the delusions, precede the difficulty in walking, or the reverse. This question has not been definitely settled, and does not intimately concern you as students. In my own experience I have generally found the mental symptoms to be first developed, and afterwards the paralytic phenomena producing difficulty in articulation and motion. This impediment in speech is more readily observed when the patient is engaged in animated conversation, and the gait is very similar to that of progressive locomotor ataxia. There seems to be a similar want of muscular co-ordination: the patient does not drag his feet as in hemiplegia, but staggers, and appears to be in danger of falling. An unequal dilatation of the pupils, without being a constant symptom, is rarely absent. Epileptiform and apoplectiform attacks are frequent.

Folsom observes, "I have usually found, when I had opportunities to investigate, that in the history of general paralysis the prodromal period, although not at the time considered important as such, is remembered as having existed. But, of the very large number of cases which I have seen in the last ten years presenting symptoms of cerebral asthenia or general neurasthenia, I have not found, even in the many who neglected treatment, a single case

of general paralysis follow ; and in the three or four cases where I ventured to provisionally make that diagnosis, either I was mistaken or a recovery followed with but very little treatment but rest. Of numerous cases with the symptoms of cerebral hyperæmia which are so common in brain-tire from over-strain, I have not seen one develop into general paralysis, nor have I known a case of general paralysis with such antecedent. In the prodromal period the best that we can do, until our means of diagnosis vastly improve, is to indicate a certain danger signal by which to warn our patients.

“When to vaso-motor indications, whether slight or not, symmetrical motor symptoms are added, the initial stage begins, and its appearance is most insidious. There is a little general muscular weakness, with some failure in concentration and adjusting skill. The occasional lapses from a former standard of living, of self-respect, and perhaps decency or honor, if they occur, are regarded as ethical rather than as requiring medical advice. The inexplicable change in personality, in character and conduct, is simply not explained. The diminished physical power or endurance is thought fatigue. The muscular incoördination and embarrassment of speech are so slight as to rarely admit of easy detection. The initial signs of general paralysis are of brain-failure. They may be recognized in a large proportion of cases, at least in those persons whose muscles and brains are highly organized. In professional and business men a less degree of impairment is recognizable than in mechanics ; in routine employments a large degree of deterioration may be unnoticed. In day-laborers an early diagnosis is simply impossible.” *

Myosis is a most common symptom : in fact, the pupils are so small, Seguin says, “as to deserve the appellation of pin-head pupils ; shading the eyes or placing the patient in a dark room causes no expansion. They are in a state of spasm, as German writers call it.”

Inequality of the pupils is almost always present. Optic neuritis is sometimes observed.

The patient's manner is often confused ; from my own experience I can fully confirm Seguin's graphic description of it:

* Folsom, Some Points regarding General Paralysis, May, 1891.

"The patient seems confused in taking a chair, or trying to leave the room ; he looks at various objects in your office regardless of the fact that you are questioning him ; he looks from his friends to you in a stupid, helpless manner, and tolerates that they answer for him. Frequently he interrupts his friends to deny the symptoms they relate. In showing his tongue he opens his mouth enormously, and makes an extensive absurd grimace. When once seen, it is a behavior never to be forgotten." Seguin also remarks, "Two years ago I saw a perfectly lucid patient presenting tremors, imperfect speech, unequal pupils, conscious failure of memory, etc. On taking leave of him he put the fee, which his wife had handed him to give me, into his own pocket, and at the door shook hands with her. The poor fellow, who has since died, was instantly aware of these errors, and laughed heartily at them."

In explanation of the psychological mechanism of these symptoms, the last-named author observes that the patient's perceptions are fleeting and imperfect, his will-power diminished, his power of attention impaired, and that he labors under a sense of unreality and uncertainty.

Regarding the optimistic delusions, too much importance should not be attached to them ; in rare instances they may never appear, and certainly they should not be considered as pathognomonic. It should also be remembered that maniacal excitement, epileptiform and apoplectiform seizures, are usually developed only in the more advanced stages of the disease.

Reflexes are generally exaggerated.

I herewith present a synopsis of symptoms from which, Seguin maintains, "an *early* diagnosis of dementia paralytica can be made by the practitioner ; nay, should be made by him.

(1) "A positive diagnosis can be made, I believe, from the speech alone ; but perhaps it is too much to ask the general practitioner to risk so much on one symptom. Impaired speech, with unequal motionless pupils, high reflexes, and slight mental symptoms, should, however, oblige the physician to make a diagnosis and remove the patient from business.

(2) "Fixed, small or unequal pupils, with changes in character, increased reflexes, and confusion in manner, should lead to a suspicion of dementia paralytica. Even the small fixed pupils alone

should, I think, excite suspicion, and lead to careful observation of the patient.

(3) "Mental slowness and inaccuracy, with any one of the other symptoms referred to, should cause a strong suspicion of incipient 'paresis.' The same is true of the inexplicable changes in the moral character of a subject above twenty years of age.

(4) "Dementia paralytica is, I might add, much more frequent among women than is generally held by authorities. They can more easily cover up signs of mental failure, and they seldom exhibit exaltation. Guided by the points I have given as of great diagnostic value, you will be able to recognize a good many female cases.

(5) "A general character of great value is the gradual slow onset of symptoms. When an adult rapidly becomes demented (foolish in manner, inattentive to his person, even to the point of not controlling his evacuations), has unequal pupils, and large quasi-choreic ataxic tremors with early convulsive seizures, it is possible that the case is one of cerebral syphilis, which may be cured by heroic treatment.

(6) "You should not be discouraged in your diagnosis by an apparent return to health after a few months, because extraordinary remissions, lasting several months, occur in the course of dementia paralytica, yet even in these remissions a critical examination almost always reveals traces of the physical symptoms."

In the last point noted, Seguin is referring to the wonderful *remissions* in the physical and mental symptoms which constitute so marked a feature in many cases of this disease. These remissions may last from six months to a year or two. Most of the reported cures of general paralysis are nothing but remissions of this disease, by which the most experienced observer may be misled.

The physical symptoms of general paresis consist in impaired control of the muscular system, diminished power of coördination, ending sooner or later in complete paralysis.

The muscles of the eye, of the fingers, of articulation, first exhibit the unmistakable evidences of progressive ataxic impairment. The gait of the patient soon becomes affected; disturbances of locomotion from want of muscular coördination are but too

apparent, and are sometimes associated with malaise and muscular pain. The patient frequently attracts attention by a staggering gait. *Many authors have observed the close relationship in some symptoms between locomotor ataxia and dementia paralytica.* The muscular movements are tremulous and uncertain; the strength is more or less diminished, and may be particularly manifested in spurts of very unequal duration. The handwriting becomes very tremulous and illegible; the articulation becomes thicker, more stammering, and unintelligible; the gait more shuffling, more staggering, and straddling.

Folsom truly observes (after describing the physical conditions which I have just quoted) that "the voice, for instance, may be loud and forcible, but the coördination sufficient for only a short explosive utterance of one syllable, and then quite an interval elapses before force can be concentrated for the next. Progressive muscular paresis becomes, finally, absolute paralysis."

General paresis, as far as heredity is concerned, has not such close relations with mental diseases as insanity in general. Folsom states that "of cases of general paralysis without a previous history of syphilis (and the same statement is true in a less degree of persons who have had syphilis) the vast majority occur in families in which there have been cases of insanity, epilepsy, or apoplexy." It is found particularly among brain-workers, and we agree with Folsom that it is not only most frequent in the stronger sex, but selects strong individuals in the prime of life, between the ages of thirty-five and fifty. Alcoholic and sexual excesses are considered very common causes. Mental shocks and emotional strain have their influence as etiological factors.

Folsom, in a monograph on "The Early Stage of General Paralysis, 1889," states that from five per cent. to nearly one hundred per cent. of general paralytics are reported by various observers as having previously had syphilis. In Mendel's cases of general paralysis the percentage of syphilitics was seventy-five.

Folsom concludes that at least two-thirds of general paralytics have previously had syphilis: he continues, "If we assume, therefore, that one person in ten in a certain adult male community (from the age of twenty-one upward) has had syphilis, it appears that an individual having ever suffered from that disease has not far from twenty times as many chances of becoming a general

paralytic as the rest of the community." He further observes that there are two reasons why general paralysis cannot be accepted as being a stage of syphilis,—first, because the usual remedies for syphilis are not of the slightest benefit, and, secondly, because there is no known method of certainly differentiating, either clinically or post mortem, cases with a previous history of syphilis from cases without it.

As regards the diagnosis of general paralysis, I again refer you to Seguin's admirable synopsis of diagnostic facts, to which may be added Folsom's statement, that "the earliest signs of general paralysis are of the slightest possible brain-failure; that the very essence and nature of general paralysis imply and involve mental symptoms in some degree, and some motor impairment, however slight, even if only judged by the test of a minute examination of what the patient can do, and how well or how ill he does it; the change in personal traits or character, and the peculiar, apathetic, indifferent, unconscious quality of the mental impairment, in uncomplicated cases, are unlike anything else."

We cannot agree with Folsom, however, as opposed to what has already been quoted from Seguin's graphic description, that "the speech may be not noticeably affected to the family, and may be only like that of a person with lips chilled by the frost or slightly under the influence of wine."

The *prognosis* of this implacable affection may be summed up in the word *fatality*.

Folsom states it in these words: "From the galloping cases of a couple of months to those slowly advancing, with long remissions, over twenty years, the average, including prodromal period, is probably not far from five (perhaps six) years. Collected from asylum statistics it is given as from two to three years."

PATHOLOGY AND MORBID ANATOMY.

Meynert asserts that preceding and causing the diffused cortical encephalitis there is a functional vaso-motor disorder which he considers curable.

"General paralysis of the insane is, according to Mendel, following Rokitansky's idea, a connective-tissue disease, affecting the nerve-cells and tissues secondarily, while Tuczek and Wernicke think that the primary disease is of the nerve-elements,—a

diffused interstitial cortical encephalitis on the one hand, or a diffused parenchymatous cortical encephalitis on the other. There is also, in well-marked cases, atrophy of the white substance, due, according to the general opinion of pathologists, to primary interstitial encephalitis ending in sclerosis." (Folsom.)

"In the majority of cases there is pachymeningitis,—also leptomeningitis, with adhesions to the cortex, especially of the anterior and antero-lateral portions, so firm that the arachnoid cannot be removed without tearing off portions of the brain; but it is sometimes scarcely observed, and may be no more than is found in persons dying simply of old age. The pia may be thickened at certain points, opaque, and without adhesions. Ependymitis is usual." (Folsom.)

As we have long known, lead-poisoning often attacks the central nervous system, and results in epileptiform attacks, mental impairment, muscular weakness, and paralysis. Some very graphic descriptions of this morbid condition have been contributed by Dr. Todd ("Clinical Lectures on Paralysis, Disease of the Brain, and other Affections of the Nervous System," published in 1855), and also by many more recent writers, especially by Folsom in his article on "Mental Diseases," in Pepper's "American System of Medicine."

This last writer states that "chronic endarteritis, arterio-sclerosis, atheroma of the cerebral arteries, may be so diffused as to simulate general paralysis, especially in drunkards and syphilitics, but the symptoms do not advance in the manner characteristic of that disease."

Referring to the fact that "multiple cerebro-spinal sclerosis of the descending form may sometimes be confounded with general paralysis while the symptoms are obscure," he especially cautions physicians against mistaking the symptoms of lead toxæmia for those of the early stage of general paralysis, directing attention, moreover, to the absence of ataxic symptoms in lead-poisoning and their regular progress in the latter form of disease.

In cases of lead paralysis, he calls attention to the importance of ascertaining the presence of lead in the urine, and to the marked improvement from the use of iodide of potassium, tonics, and electricity.

I would add that the history of the case, the existence of prior

attacks of *colica pictonum*, and the presence of the "gingival line" are points not to be overlooked in the differential diagnosis.

Folsom appropriately calls attention to the fact that "chronic and persistent alcoholism is always attended with some mental impairment, which may so resemble the dementia of general paralysis, with marked moral perversion, mental exaltation, grand delusions, muscular tremor, ataxic symptoms, and impaired muscular power, as to make the diagnosis doubtful for several months, until removal of the cause (alcohol) in the course of time causes the symptoms to so abate as to make the real character of the disease evident."

TREATMENT.

Rest and general hygienic measures should be enforced. Ergot and the careful use of the bromides may be of some slight use and transient effect in the earlier stages. Enemas and laxatives, especially *rhamnus frangula*, should be resorted to when the bowels are constipated. Chloral and the bromides are of great service in controlling the epileptiform attacks. Conium, hyoscyamine, and hydrobromate of hyoscyne may be judiciously used to control the maniacal exacerbations. Iodide of potassium has its advocates. Many of these patients must necessarily be sent to asylums, as they may at any moment become very dangerous. The treatment of general paralysis is at best most unsatisfactory.

Ergotinine in general paralysis.—Several authors refer to ergot in the treatment of general paralysis, but Christian reports quite marked success in checking the convulsive seizures of this disease by hypodermatic injections of ergotinine. He employs one-sixtieth of a grain (0.001 gramme) in solution, and has found one or two injections sufficient to check the convulsions.*

In cases with syphilitic histories, it is well to resort to specific treatment and give the patient the benefit of any doubt. Iodide of potassium in heroic doses, and mercurials, should be essayed. A combination of half a grain of protiodide of mercury with a quarter of a grain of extract of opium, as suggested by my friend Dr. W. A. Hardaway, of this city, I have found very serviceable in many syphilitic affections of the nervous system, given morning and evening.

* Brush, *Annual of the Universal Medical Sciences*, Sajous, 1891.

SURGICAL INTERFERENCE IN GENERAL PARALYSIS.

"A recent issue of the *American Journal of Psychology* contains a number of papers discussing the question of surgical interference in general paralysis. It is stated that to Dr. T. C. Shaw belongs the credit of making the first attempt, surgically, to alter the course of this disease. To his mind the pathological appearances in general paralysis pointed to an irritative, probably inflammatory, process in the upper layers of the convolutions. The theory of the operation was that, by producing an alteration in the existing state of the morbid process, a new and nutritive process might be set up. On the theory of nerve-stretching, he proposed to stretch the brain, by giving it more space in which to expand, allowing it to relieve itself of the increased arterial pressure shown by the sphygmograph to be one of the early conditions of general paralysis. Shaw considers that the operation in his case was justified by the success attending it, as the patient's general condition improved, although the prominent bulbar symptoms remained. Dr. Batty Tuke's patient also improved for a short time after the operation, but then relapsed. His case was further advanced than Shaw's, but he felt that the results had warranted the operation. The pressure theory, according to Tuke, makes it certain that obstructed lymph may make its way but imperfectly by natural channels to the pia-matral space, and become diffused through the tissues, injuring and displacing cells and fibres and impairing their functional activity. The operation, by permitting a healthy action of the lymphatics and blood-vessels, stays the process of sclerosis.

"Rivington, however, considers that the entire mass of pathological evidence is absolutely contradictory of such a theory as this, and that the typical cell degenerations found in general paralysis are not such as may be expected to follow simple excess of fluid-pressure, but are rather true degenerations, due to acute interstitial anomalies, with no notable differences between the changes through which the cells pass and those in senile atrophy, and that there is no excess of fluid in the first stage, while the second stage is one of extraordinary development of the lymph connective system of the brain with a parallel degeneration and disappearance of nerve-elements, the axis-cylinders of which are

denuded. In the first stage, then, the only one in which an operation would be justifiable, there is no excess of fluid, and in no stage is the fluid of more than secondary importance. The final deductions from a survey of the evidence in these cases are that the pathology upon which the operations were founded is opposed to all the best knowledge on the subject, and that the collation of two cases warrants nothing so clearly as the opinion that little good can be expected from the operation of trephining in general paralysis.

"In this connection it may be mentioned that a number of cases have been reported by Burckhardt in which excision of portions of the cerebral cortex was performed upon the insane; it would appear that some improvement was thus obtained in the quieting of excitement and in the suppression of hallucinations.

"Tuke discusses the surgical treatment of intra-cranial fluid-pressure, which he thinks is not awarded its true value. He gives two instances of 'general paralysis of the insane,' one of his own and one of Claye Shaw's, in which trephining was followed by marked improvement, and suggests that there may be other cases, characterized by coma, delirium, and acute mania, produced by a congested and dropsical condition of the brain, or by meningitis, in which an opening into the dura would be of advantage. He thinks that the importance of the lymphatic system of the encephalon, as a factor in morbid processes, has been too much overlooked.

"In Shaw's case there was a history of severe injury above the left ear fifteen months previously. After trephining, the patient, a man aged twenty-nine, improved in many respects, mentally as well as physically, but six months later he died after prolonged coma preceded by strong convulsions. Cripps, who was the operator in this case, states that the treatment was adopted with the view of relieving undue pressure on the brain caused by excess of fluid within the cranium.

"Rivington has opposed the views of Tuke and Shaw on pathological and mechanical grounds. He does not think that there is warrant for expectation of benefit from operative interference in cases of general paralysis of the insane.

"In another case of general paresis trephining is reported by Wagner to have produced marked improvement. But it seems

likely that in this instance, as in some, at least, of those published heretofore, there were pressure-symptoms superadded to those of general paresis; these being relieved by operation, there was, incidentally, an improvement in the patient's main disorder. One can hardly believe that it will be found possible to remedy a condition of degeneration, such as exists in the general paralysis of the insane, by any surgical procedure." *

"This subject is discussed by Percy Smith, and attention is called to the fact that it is not uncommon for long periods of remission, or for real or supposed recovery, to be observed in cases of general paresis. Further, he quotes from Mickle the statement that several instances of recovery or of very prolonged remission have supervened on accidents, violent injuries, or diseases of such a kind as to produce so-called 'revulsive effects.' " †

* Packard, *Annual of the Universal Medical Sciences*, Sajous, 1891.

† *Annual of the Universal Medical Sciences*, Sajous, 1891.

LECTURE XIX.

DIAGNOSIS OF INSANITY IN GENERAL.

Diagnosis of Insanity in General—Cautions necessary in Determining Insanity—Simulated Insanity—Tests—Simulation of Insanity by the Insane—Prognosis of Insanity—Pathology and Anatomical Appearances of Insanity—The Frontal Lobes are the Seat of Intellect—Lesions of Frontal Lobes produce Changes of Character—Recapitulation—Brain-Surgery in Insanity—Comparative Advantages of Home and Asylum Treatment.

GENTLEMEN,—A few brief remarks in regard to the diagnosis of insanity will now be appropriate. Is there any other affection with which it may be confounded? Is it not possible to make a mistake? I believe that, when fully developed, it can be easily recognized with ordinary care and scrutiny. The difficulty is far greater when insanity is only *suspected*, especially when you are called upon as expert witnesses in a court of law. Some diseases might be confounded with it. Acute meningitis with acute delirium has been mistaken for acute mania; but in the latter disease there is no elevation of temperature, which is a marked feature of the former; febrile phenomena exist in all inflammatory affections, and are therefore present in meningeal disorders. There is, it is true, an elevation of temperature in one form of insanity, already described, but only in one,—acute delirious mania.

Delirium tremens presents some of the features of insanity which are manifested when a person has been habitually addicted to alcoholic excesses and prolonged debauches. In ordinary cases of delirium tremens it is hardly possible to make a mistake; because, as you will learn more particularly when we study that disease, there always exists a peculiar loquacity, a remarkable, good-natured delirium, with *visual* hallucinations, enabling a physician of only slight experience to make the distinction. The tremor also is peculiar, produced, as it is, by the action of the alcohol upon the motor nervous system. But a man may be deeply under alcoholic influence and extremely excited, perhaps

showing strong homicidal or suicidal tendencies, while the physician may be unable to say whether he is laboring under acute mania or alcoholism. The diagnosis at times may be quite difficult ; but we have one fact that may help us in such cases, which is, that in ordinary alcoholism the patient is much better after good and refreshing sleep. If, therefore, after having obtained the desired rest, the patient awakes as wild and violent as he was on the day previous, or even more so, the probabilities are that it is a case of acute mania instead of delirium tremens. Besides this, the absence of the ordinary symptoms of alcoholism—namely, the clammy perspiration, the characteristic visual hallucinations, the rapid, frequent pulse, and the tremor—readily removes many of the difficulties attending the diagnosis.

To distinguish some particular forms of insanity, and to determine the fact of a man's being *sane* or *insane*, are wholly different questions, which are apt to prove very vexatious. Should you declare a sane person to be a lunatic, and send him to an asylum, thereby unjustly depriving him of his liberty, you may expect that, if he gets out and proves the error of your diagnosis, he will cause you much trouble, especially if you possess any property which would justify him in instituting civil proceedings against you. Now, how are you to determine these matters with safety, as there is so much difference between the cases we meet ? One general rule I can give you in this regard ; which is, always to avoid rash and hasty action : never to hurry your decision, but to study the history of the patient, his idiosyncrasies, and in particular any change in his character,—an important matter, to which I have frequently adverted throughout my lectures upon insanity. This departure from one's normal self is manifested by certain actions or feelings which never before existed. Be cautious, and do not blindly accept the statements of relatives, as they may be biassed by a desire to remove the person out of their way. You cannot be too prudent ; and you should always *give the patient the benefit of the doubt*. Never blindly assume responsibilities : if the patient be really insane, he will sooner or later show it. If you have carefully followed me in these lectures, you will know that, although a man may suffer from derangement of the affective type with very little evidence of accompanying ideational insanity,—speaking quite coherently and rationally,—he, nevertheless, *cannot control*

his actions ; and, as this affective form underlies all others, his actions at some time or other are sure to reveal his insanity. If this be the case, the patient must, of course, be isolated in an asylum, as he is out of harmony with his social sphere, and must be sequestered to prevent him from exercising a pernicious influence on the individuals composing the society of which he is an element. I repeat it, be very cautious and prudent, before giving an opinion in the affirmative and participating in the subsequent proceedings ; for, if you do not, you may bring upon yourselves an immense amount of trouble.

I shall not dwell long upon the differential diagnosis between mania, melancholia, and the other forms of insanity. I only wish once more to impress upon your minds that all insane persons do not necessarily manifest their insanity as plainly as the laity generally imagine, and that even experts, after numerous conversations, may be misled, if not upon their guard. If experts have to exercise care, how much more does it behoove the general practitioner to avoid all possible sources of fallacy ! If you remember this, you may yet thank me for the stress I lay upon these facts, whose appreciation may save many a physician from most perplexing annoyances.

If, as sometimes happens, a delusion exists, and the patient endeavors to deceive you, it may require many visits before you can arrive at a knowledge of the true nature of the case, and in matters of this kind you should never allow yourself to be influenced by the desire of friends so as thereby to be placed in a false position. I have already told you of a case in which an individual so carefully concealed his insanity that, after having him under my care for two years, I felt convinced of his sanity, and consequently wrote to his brother to come and take him home ; yet on the very night of the brother's arrival the patient became so violent and aggressive as to necessitate his immediate restraint. Of course such people are harmless in an asylum ; but where they are at large, homicidal tendencies very frequently manifest themselves, as well as other dangerous propensities.

A word concerning *assumed* insanity. You may be called upon to give your opinion in a trial where a man affects insanity in order to evade punishment. You may not have the opportunity of calling in experts, and the criminal will endeavor to deceive

you ; therefore you must be on your guard. Recollect that no man can for any great length of time act the part of a maniac. He cannot feign insomnia ; for if you watch him closely you will find that in moments of exhaustion he will sleep soundly without the use of an anodyne. Sometimes such people purposely refuse medication. If you have ever seen such cases, you will readily recognize the fact that pretended maniacs invariably *overact* their parts.

In commenting upon legitimate devices for exposing the simulation of insanity, Spitzka makes the following observations : " When examining the patient, let the interlocutor remark in an undertone to a by-stander that if such and such a sign were present he would know in which ward to put him, or under which form of insanity to classify the subject. This is far safer than the suggestion adopted from the French writers by Ray, and copied from him by some recent pamphleteers, of saying that if such and such a sign were present the interlocutor would believe the man to be *insane*. This would put a cunning simulator on his guard. The writer had to deal with such a one in the case of a child-abductor who had feigned insanity in a jail once before. Suspecting that the recommendation of the older writers would have failed, the writer turned to a by-stander and said, ' This is a most interesting case, and I have frequently remarked that these patients do not remember what city they are from.' The criminal had previously assigned Baltimore as his home, and this was, according to the legal papers in the case, correct ; but on being interrogated he said, in a hesitating and whining voice altogether unnatural to a person suffering from monomania with sexual perversion (the form claimed to exist), '*Concord, Cincinnati.*' "

Dr. C. H. Hughes, of St. Louis, has written an excellent article upon " Simulation of Insanity by the Insane," proving that " the insane appear at times, when they have an object to accomplish, more crazy than and different from what they really are ; this is the sense in which we use the term simulation, and this condition is akin to that of feigning by the sane. Simulation, while it presupposes a degree of sanity, does not require that the patients should be wholly sound in mind, and it might be attempted by a convalescent patient not thoroughly recovered, or desirous of remaining longer in the hospital, or for some other cause."

In melancholia, which is a deep derangement of the affective type, we know that, in consequence of a disinclination to take food, the patient will resist its administration. If chronic mania should be simulated, you might be easily misled. But there is one peculiarity in all these simulations of insanity, which the books refer to, that those who attempt the deception always appear to be devoid of memory. They invariably fail to have any recollection or appreciation of time, locality, or condition. These are symptoms more characteristic of softening of the brain than of insanity. I have already told you, in speaking of partial cerebral anæmia, that there is a loss of memory, which becomes more apparent as the disease progresses. In insanity, on the other hand, the most remarkable memory is often manifested, except in dementia, which, of course, is readily recognized by the expression, or, more correctly, the want of expression, of the face, and by the almost total abolition of the mental faculties, with a previous history of some acute form of insanity. But in feigning, these people will often affect to forget that they saw you the day before, or fail to recollect their own names, or those even of their nearest relatives, etc. We know the absurdity of such assumptions, a knowledge of incalculable benefit to the diagnostician. Recollect also one other important fact in this connection. In some persons insanity is easily recognizable, while in others it is not so, nay, it may be almost impossible to affirm its presence,—even to such an extent that experts will be unsuccessful. I recall the case of a man who was confined in St. Vincent's Asylum by an order of the judge of the criminal court, so that through careful observation I might be enabled to determine the presence or absence of insanity. After studying his case for three months I was unable to decide, when he was sent back to jail and subsequently admitted simulation.

The prognosis can be summed up in a few words. Insanity is curable in direct ratio with its duration. If acute, it is often amenable to treatment; but not so if chronic. Therefore, *when of less than three months' duration*, it may (in at least seventy per cent. of cases) be easily conquered by therapeutic measures; but when it passes this limit a cure becomes more difficult as its duration is greater. Again, in patients not over thirty years of age, insanity is quite manageable, but in old people the prognosis

is very unfavorable. Where the insane temperament exists, or where insanity has existed in the family, there is less hope of *permanent* cure. Some of these cases may recover, but in those who are the victims of an innate vice of constitution or of an unstable nervous organization a relapse may be confidently predicted. Such individuals remain always liable to outbursts of insanity, and to have repeated attacks. If you know the histories of cases in asylums, you may easily predict that certain persons will soon return when you discharge them after apparent cure. The relapse may be postponed, but you may rest assured that it will occur sooner or later.

PATHOLOGY AND ANATOMICAL APPEARANCES.

I will now make a few general remarks in regard to the *pathology* of insanity. In my lectures upon its different forms I have so frequently referred to its underlying pathology that there is but little left to add. I have already told you that insanity is a disease of the brain, having its seat in definite parts of that organ,—in the cortical portions of the convolutions of the cerebral hemispheres,—though its initiatory cause may not exist there, but may have a remote seat, as the bladder, uterus, lungs, heart, or some other organ. But the proximate seat, the anatomical *site* of insanity, is always the brain. If this be so, do you expect always to find manifest changes in the brain after death from insanity? No; for, though it may have been caused by reflex irritation, as from the uterus, for instance, propagated to the brain, still, anatomical change may not be apparent either to the unassisted eye or even to the higher powers of the microscope. The fact, nevertheless, that we cannot ascertain such changes is surely no proof that they do not exist. Consider now that we are dealing here with anatomical elements of wonderful subtlety,—with a labyrinth of untrodden intricacy,—indeed, with the parts which evolve and elaborate thought itself. We have no appliances sufficiently subtle to detect all the molecular changes which may have occurred in this most delicate of organized structures,—the brain. It may be diseased, and its disease still escape the scrutiny of our methods of investigation. In order that morbid intellectual phenomena may be produced, we must presuppose an alteration in the physiological condition of the cortical cells of the cerebral

convolutions, or at least in those that preside over the elaboration of mental phenomena.

"Welt, as the result of careful studies upon eight cases of injury or disease of the *frontal lobes*, under his personal observation, in all of which autopsies were made, concludes that such *changes in character* and disposition as very frequently occur after lesions of the *anterior lobe*, directly result from such injuries or diseases. These alterations of character and disposition are frequently the only symptoms, no motor or sensory disturbances being evident. In all of the eight autopsies, the convex cortical surface of the frontal lobe was the portion affected. In three of these cases the lesion involved the first convolution alone, in three others the first and third were involved, and in two the lesion extended from the first to the third and included the second. These results, together with facts accumulated from an analysis of autopsies in a large number of similar cases reported by others, showed that the region most uniformly affected was that nearest the median line or the first frontal, and of the right rather than the left. The mental changes vary greatly as to the time of onset, nature, and duration. It will be seen that these conclusions are in accord with Ferrier's experiments upon this region in monkeys, and those of Goltz upon dogs, and of Luciani upon pigeons and dogs. In another part of the *Annual* will be found a synopsis of three cases reported by Thompson, of tumors located in the frontal region, having an important bearing upon the functions of this portion of the brain.

"A typical example of the physical and mental effects of injury to the fore brain is furnished in the history of an inmate of the Zurich Hospital. The man, while drunk, fell one hundred feet, suffering a splinter fracture of both frontal bones, with contusion and efflux of brain-substance from the frontal regions. The man, who had before been peaceable, good-natured, cheerful, and cleanly, became malicious, slanderous, violently quarrelsome, and dirty. No other morbid changes were manifested." *

The late Dr. Gray, of the Utica Asylum, one of the most gifted and distinguished of American alienists, has greatly contributed to

* Landon Carter Gray, *Annual of the Universal Medical Sciences*, Sajous, 1891.

our knowledge of insanity, and has illustrated his contributions to cerebral pathology by means of beautiful micro-photographs. His conclusions, to quote his own words, are, "Although the cases thus far examined may be regarded insufficient to establish general conclusions, they go to strengthen the conviction sustained by the laws of general pathology, that insanity is a physical disease of the brain, and that the mental phenomena are symptoms; further, that the microscope, with patient and close investigation, will continue to disclose structural changes in the cerebral tissue as marked as those heretofore unsuspected when examinations were limited to the scalpel and naked eye; and in these investigations, when the entire range of the disease, in every stage of its progress, shall have been brought under the microscope, we may be able to solve the problem of the morbid processes conveniently denominated insanity.

"Another conclusion to which these investigations must naturally lead is, that the variety and changes in the predominant symptoms of insanity may find their explanation not so much in the variety of lesions as in the special parts of the cerebral centres which are morbidly involved in each case; or, to bring the idea within narrower limits, that ideational, emotional, and motor disturbances have their foundation in the extent and degree to which the nerve-elements that minister to the execution of the intellectual and motor acts are involved in the lesion. When the disease reaches its ultimate stage, all distinctions cease, dementia being the same closing stage of every so-called form of insanity." In the Thirty-Second Annual Report of the State Lunatic Asylum, Dr. Gray remarks, "Continued experience not only confirms the truth of this position, but also that, in all cases, post-mortem examination will reveal organic lesions, changes in the condition of the vessels or structures of the brain or its membranes." He discards the term functional disease as a "phantom of the mind," as it has been designated by Winslow.*

According to the older *humoral* or *vascular* theory, all forms of insanity were considered as the result of congestion of some part of the brain. But just as in inflammation there is always a primary tissue-irritation, which is extra-vascular, or, I may say,

* American Journal of Insanity, July, 1875.

interstitial, so insanity originates from disturbances, whether primarily interstitial or parenchymatous, in the ultimate nervous constituents outside of the vessels, the primary changes not being due to an increased flow of blood. I care not what doctrine of inflammation you have learned, I hold that since the views of Virchow have been promulgated it must be held as established that in inflammation there is an irritation of the cells composing the tissue outside of the vessels. Exactly the same law holds good in insanity, although itself not originally an inflammatory disease, for Dr. Gray has proved that increased connective-tissue proliferation plays an important part as one of its primary factors. It is true that insanity may be induced by some adynamic states of the system, through influences of the blood on the cells and ultimate constituents of the cerebral nervous texture; but proneness to this disease does not reside in the blood itself, but is founded in a peculiar instability of the cerebral cells, composing as they do the material structure of the temple in which the mind resides. Whenever perturbing influences are experienced by the material fabric (the bricks and mortar of which the temple is composed), disturbances of the normal evolution of mental phenomena will necessarily occur, improper ideation will ensue, and insanity be developed. Therefore the starting-point of all forms of insanity is in the fundamental constituent elements or cells of the cortical substance of the hemispheres of the brain. Here the disease starts *ab initio*. Hence you readily see that it is the constituent elements of the cells which are at fault in insanity. These cells may have their own laws of elaboration and evolution, of which we know nothing, and which science may perhaps never reveal. But how is it with other cells? Do we exactly know the working of any of them? Though insanity probably starts in the cerebral cells, it may not be at all improbable that there are consequent though undetectable changes in every other cell in the body, so that an insane person may be insane to the very tips of his fingers. This is surely no more difficult to understand than the fact that the renal or hepatic cells never fail to select those constituents of the blood which are adapted to the elaboration of their proper secretions. Can we explain how aliments are changed into flesh? We know that they are so changed, and accept the undeniable fact of daily transubstantiation during

digestion ; but can we understand all the special transformations which occur in these processes of assimilation ? Or can we account for cell-impregnation ? Why is it that when a woman has children by a second husband they may bear a very remarkable resemblance to the first, though he may have been dead for years ? We know that it is so, and might offer an explanation by saying that the cells had been perfused with a certain psychical influence lasting for a lifetime ; but such an explanation would be at best hypothetical.

But though some of the pathological conditions of insanity are very obscure, there are others that we can more readily understand. For instance, to have a healthy mind, one must have a healthy body. If the body be pervaded with poisoned blood, whether by the effects of syphilis, of typhoid fever, or of variola, etc., it is evident that the functions of the brain will be more or less at fault, because for the healthy evolution of cerebral phenomena healthy blood—normal, both quantitatively and qualitatively—is indispensable. You must recollect that a man may be insane with either an anæmic or a hyperæmic condition of the brain. Poisoned blood cannot subserve the proper action of the ideational centres. Healthy blood is required for the evolution of normal thought, just as in the liver pure blood is requisite for the production of normal bile. You may, perhaps, accuse me of holding materialistic views, because I compare the psychical evolution to that of bile. I must state, however, that the more I study the phenomena of life and the functions of our organism, especially those of the brain, with its wonderful ramifications through the system, its ever-varying and almost limitless psychological powers, its delicate mechanism, and the mystery of its functions, the more I appreciate the difficulties of cerebral physiology and psychology. The brain is like an æolian harp, whose strings vibrating to the passing wind give forth euphonious sounds. The mind employs the brain for the production of thought. The mind is not material ; but if the brain be not in a physiological condition, it becomes a false messenger, an untruthful interpreter of external things, and its play is inconsonant, inharmonious, discordant, and what we call insanity appears. That the brain is only the physical organ of the mind is shown by the fact that the quantity of the phosphates in the urine varies with the amount of mental

labor undergone ; for the more violent the action of the mind, the greater is the destruction, or the retrograde metamorphosis, of the brain-tissue.

So much for the manner in which a change in quantity or quality of the blood may produce insanity. A few points still claim our consideration.

We have reason to believe that insanity originates in the tissues outside of the vessels ; it may, however, be influenced by vascular causes, and especially by reflex action. I do not think I could give a more practical illustration of this than by citing a case already mentioned, one of prolapsus uteri attended by melancholia, where the melancholia disappeared immediately upon the reduction of the prolapsed organ and its restoration to its normal position ; but as soon as the pessary was removed and the uterus once more descended, the insanity reappeared, to vanish again upon a subsequent reduction of the prolapsus. I could not give you a plainer illustration of the effects of reflex action. It is not more difficult to understand than that dizziness and dilatation of the pupil are often symptomatic of *tænia* and disappear with the expulsion of the parasite. This proves the necessity of ascertaining the cause of the reflex action, whether it be thoracic, pelvic, or abdominal. Direct your remedies to the causative condition, and in relieving this you will often eradicate the insanity.

It is quite a common thing to find insane females suffering from metritis, cervicitis, etc. ; and the cure of these complications has often restored them to reason. I recollect a case where all previous treatment had failed, in which after the cure of an obstinate leucorrhœa the patient showed the first symptoms of improvement, the insanity finally disappearing. This shows the connection between cause and effect ; and as it is in insanity, so it is sometimes in paraplegia caused by reflex irritation from disease of the genito-urinary organs, which is occasionally cured by removing a simple gonorrhœa. If such an affection can have these effects upon the spinal cord, how subservient to the influence of reflexive irritation must we concede the brain to be,—this great centre, this metropolis of the body, in relation with every muscle, with every nerve and fibre of our system !

“ Indeed, nearly every pathological condition of the brain known in insanity, in kind, if not in extent and degree, may be found in

diseased or injured brains where there has been no mental disease in consequence. . . .

“In those forms of mental disease where changes are found, the most important and constant are in the cortex of the brain, especially in the fore, upper, and middle parts of the periphery, involving usually also the membranes. In beginning acute mania the condition of the blood affecting the brain or the pathological changes are probably as nearly identical with those in the acute stage of pneumonia, certain forms of typhoid fever, cerebro-spinal meningitis, and other diseases, as the symptoms of the mania are now and then difficult to differentiate from those of the other diseases just mentioned. In rheumatism, syphilis, malarial poisoning, and Bright’s disease, with mania, we find no distinctive pathological conditions to account for the maniacal symptoms.

“If asked whether there is a fixed lesion of the brain or any of its parts corresponding to given psychological changes, we should be obliged to say no, except in the cases of incurable dementia. If asked whether there are important morbid changes corresponding with all cases of insanity, we can only say yes, sooner or later, in the majority of cases, and that there are certain destructive lesions, chiefly inflammatory, atrophic, and degenerative, which invariably mean marked deterioration of the mind. As regards diseases of other organs than the brain, the insane, like the sane, die of all of them, and in especially large numbers of pulmonary consumption.

“Insanity may, both in its acute and chronic forms, be the result or symptom of simple anomalous excitation or nutrition of the brain or of inhibition of some of its portions, without any change in its gross appearances which can be detected by our present methods of research. In the majority of cases there are found diseased conditions which become more manifest the longer the duration of the disease, appearing for the most part in the blood-vessels, pia mater, and cortex of the brain, but also in the medullary portion, many of which are recognized only in their late stages. In the functional mental diseases there is no characteristic lesion of the brain as yet recognizable, even in the latest stages, more than is to be found in the brains of persons dying from other causes. When apparently local injuries or diseases cause insanity, they probably do so through a general disturbance

of the brain, or through diffused disease resulting therefrom, and for the most part affecting both hemispheres. The molecular, chemical, anatomical, physiological, pathological, or physical changes in the brain which give rise to insanity, and their relation to the grosser pathological conditions of the brain, are still not clearly made out." (Folsom.)

Before dismissing the subject, I would observe that whenever a predisposition to insanity exists, it may be induced by *excessive functional action* of any part, notably of the brain itself. Such excessive functional activity will produce an irritative exhaustion, whose continued influence will result in emotional disturbance; this being reflected to the brain, insanity follows. Knowing the laws of health, we should never forget that "tired nature's sweet restorer," sleep, which permits recuperation of the forces and prevents incessant wear of the tissues, is the great prophylactic of insanity: so that whenever prolonged loss of rest exists, we must at once induce profound and adequate sleep.

RECAPITULATION.

Insanity is a disease which has always its seat, though not necessarily its cause, in the brain. It is manifested, like other maladies, in an acute and a chronic form. Its origin, I repeat, is not invariably in the brain, for in consequence of the close sympathy which exists between this great nervous centre and all the other portions of the body, morbid action starting in any part of the economy may, in susceptible individuals, be the exciting cause of a reflex irritation which disturbs the functions and impairs the delicate mechanism of that wonderful organ whence all intellectual manifestations are derived.

Too much stress cannot be laid upon the fact that insanity is curable in direct ratio with its duration. Although cases of many years' continuance are sometimes cured, and notwithstanding the well-admitted fact that no case is necessarily incurable, yet it is practically in the field of acute cases that the physician may expect to reap his most abundant and successful harvests. Many authors place the limit of the acute stage at three months from inception. The farther we pass beyond the third month, the more difficult will be the cure. You are doubtless aware that the prognostications of ordinary pulmonary affections are favorable just in

proportion to their recency or remoteness of origin. Acute inflammation of the lungs, with our modern therapeutic resources, is not often a fatal disease. Phthisis pulmonalis still but too frequently baffles our best-directed efforts to arrest its terrible ravages. On the other hand, the management of acute mania or melancholia frequently attests the brilliant results accomplished by psychological physicians, while monomania, chronic mania, and dementia are still the opprobrium of our art. A brain which is the seat of dementia is comparable to a battle-field, where all was once storm, fury, and irresistible violence; but now, wreck, ruin, and desolation. For these unfortunate victims the propitious moment has forever passed, because they did not reach the haven of an asylum in time to prevent a disaster from which they can never recover. This irreparable mischief has been inflicted upon them in consequence of the procrastination of friends, and the defects of faulty and unphilosophical legislation. The conclusion to be drawn is manifest,—all patients should be sent to an asylum upon the very first development of mental aberration; the success which will so often follow such a course will soon convince you of its wisdom. The morbid processes, if you lose valuable time, make sure and rapid strides; every day that you allow to pass without medical treatment tends to insure the terrible doom of chronic insanity which impends over these unfortunates.

Emory Lanphear, of Kansas City, Missouri, in a late article in the *American Journal of Surgery and Gynecology*, observes,—

“The suggestion to open the skull in insanity is not new, but its execution is recent. Burckhardt has detailed six cases of insanity with marked hallucinations, which he subjected to operative treatment. ‘In two cases he aimed to intersect the paths of association, which he thinks transmits the pathological impression coming from sensory parts and certain ideogenic areas of the brain; a portion of the frontal and parietal lobes, before and behind the ascending convolutions, were removed with very satisfactory results in one case, the other being still under treatment. In the other four cases the hallucinations were more or less acute, and in these cases the operator attacked the centres through whose injury sensory and motor aphasia are produced, and removed a part of the first temporal and third frontal on the left side, which appeared diseased, and with satisfactory results. It is possible

that, with additional experience and a minute study of the pathological changes seen in the brain, the knife may be the means of restoring to reason many cases now considered incurable.' But the question naturally arises, were or were not these cases the result of the operation *per se*? Only further experimentation and careful observation can determine. It is in the first stage of general paresis that I shall look for beneficial results, for here we have a mental disease (so called) which is dependent upon gross lesions. In insanity due to intra-cranial growths the indications are always to operate."

COMPARATIVE ADVANTAGES OF HOME AND ASYLUM TREATMENT.

Since the publication of the first edition of these lectures, Seguin, in his "American Clinical Lectures," has expressed views on the above subject almost identical with those which I am about to quote from the recent excellent treatise on "Familiar Forms of Nervous Disease," of M. Allen Starr. I need hardly add that, after an experience of a quarter of a century in the treatment of insanity, I can fully corroborate all his assertions.

"While it is apparent from the foregoing that I believe in the early removal of most cases of insanity from the environment in which the psychosis has developed, I am by no means an admirer of asylums in general as now conducted. The large public institutions are hampered in their treatment by the enormous number of patients and by the lack of a corresponding number of physicians and attendants. The superintendent is seldom a thoroughly trained alienist, and in some States these charities of the people are unfortunately made to subserve the interest of the great political machine. Patients cannot derive that benefit which they should from the medical superintendent's long experience, because their individual requirements must be sacrificed to the many demands upon his time in the general management. His assistants are too few and too preoccupied with their clerical and office duties to carefully individualize and treat the patients. Finally, there are too many patients. A State asylum containing but two hundred patients has but an indifferent public standing. It must have a thousand or two thousand patients in order to satisfy the ambition of the community and of the managers. Small wonder, then,

that the actual object of the institution should so often be lost to sight!

“Until, therefore, these great charities, now little more than warehouses for the storage of articles unnecessary or in society’s way, conform more to the character of a hospital, with its modern equipment, its attending physicians, and its consulting specialists, the higher classes of private asylums in the hands of men who are known for their professional attainments and probity of character will always be more desirable as places for the reception and treatment of such insane patients as are so fortunate as to be able to enjoy their advantages.

“It is to be hoped, however, that at some future day our general hospitals will provide special wards or pavilions for this particular class of cases, so that in every city one or several places will be at all times ready to receive the acute insane and care for their sick brains in the highest scientific manner.”

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